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THE
LARYNGOSCOPE.

VOL. XXXVI NOVEMBER, 1926. No. 11

ORIGINAL COMMUNICATIONS.

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PROGRESSIVE DEAFNESS. ENDOCRINE MALFUNCTION AS AN IMPORTANT ETIOLOGICAL FACTOR: ITS INCIDENCE AND TREATMENT.*

DR. CHARLES H. LAWRENCE, Boston.

A survey of the audiometer records of five hundred normal individuals** shows marked variations in hearing ability as determined by that method of measurement. Analysis of these variations indicates that the hearing acuity is less influenced by training or vocation than by the nutritional level of the individual. Thus reference to Chart No. 1 shows that the group showing the highest levels of hearing were college athletes in training. Musicians, piano tuners, telephone operators, and blind persons, all of whom are supposed to have most acute hearing, but whose physical condition was less perfect, show less perfect auditory function, while children in a "hospital school" for the undernourished show a marked depression of hearing acuity.

In view of this apparent relation between nutritional level and audtiory function, it seems worth while to report the findings in 31 cases of progressive noninfectious deafness which have been carefully studied at the Evans Memorial and which have been under treatment for from 6 to 18 months. The study of these cases included a careful history and physical examination, metabolic studies, complete blood examination, including the Wassermann reaction in

*Reprinted from Evans Memorial Publication No. 123 A 69.
**Arch. Oto-Laryn., 1-524 (1925).
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TABLE 1.

General Data.

Total Cases	31				
Female	26	—	84%		
Endocrine		21		Non-Endocrine	5
Male	5	—	16%		
Endocrine		1		Non-Endocrine	4

DURATION OF DEAFNESS	
10 years or over	6
6-10 years	6
5 "	5
4 "	6
3 "	3
2 "	6
1 "	2

ENDOCRINE CONDITIONS	
Thyroid	8
Pituitary	7
Gonad	7
NON-ENDOCRINE CONDITIONS	
Anaemia	3
Infections	6

TABLE II.

Symptoms	Endocrine.	Non-Endocrine.
Fatigability	18 — 82%	2 — 22%
Tinnitus	9 — 41%	—
Vertigo	2 — 9%	—
Blood Pressure	90/62, 95/60	115/75
	104/60, 100/66	Or higher
	etc.	

WEIGHT.	
More than 10% underweight	9
" " 10% overweight	4
Within + or — 10%	9

CATAMENIA. (Onset)		
15th year	5	
16th year	3	
17th year	4	
Total	12	
Before 15th	5	(4 of these had irregular function)
Undetermined	5	

TABLE III.

BLOOD.		
Lymphocytosis	14	
Increased NPN	1	(Thyroid)
Increased Uric Acid	3	(Pituitary)
Increased Urea	1	(Gonad)
Increased Urine Residual Nitrogen		12
Decreased Alveolar Carbon Dioxide		17

BASAL METABOLIC RATE:	
Below —25%	3
—20% to —25%	1
—15% to —20%	5
—10% to —15%	5
+10% to —10%	6
+10% to +10%	3

TABLE IV

TREATMENT

Endocrine.

Treated		Untreated
19		3
Improved	14	
Not improved	5	

DEGREE OF IMPROVEMENT.

	Thyroid	Pituitary	Gonad	Total
Marked	2	2	0	4
Definite	2	2	1	5
Slight	1	1	3	5
None	1	1	3	5

NON-ENDOCRINE.

2 Anaemias improved.

TABLE V.

ANALYSIS OF NON IMPROVED.

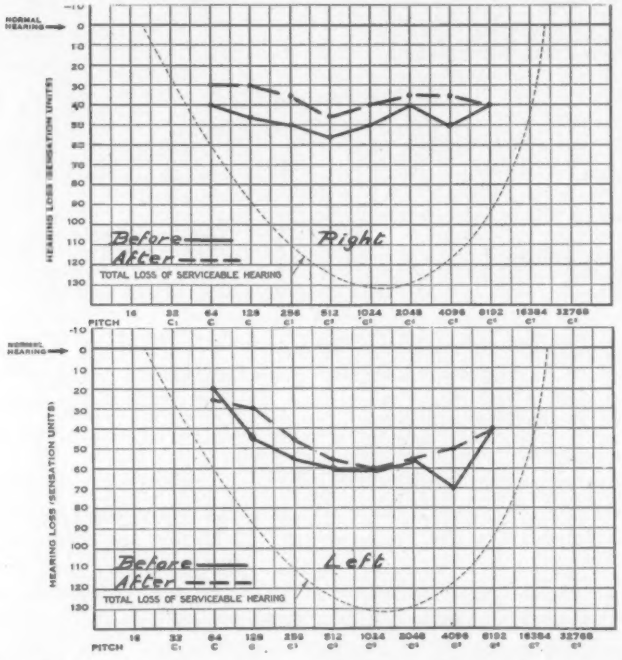
Diagnosis	Age	Duration	Remarks
Thyroid	40	7	(Protein Sensitive (Tonsils +
Gonad	46	26	—
Gonad	22	7	Treatment intermittent
Gonad	29	2	Not improved in 6 months
Pituitary	17	7	Treatment intermittent

every case, and such special examinations by ophthalmologist, Roentgenologist, or other consultant as were indicated. All the patients were referred to me by members of the staff of the Massachusetts Eye and Ear Infirmary, by whom the diagnosis of otosclerosis was established.

In none of these patients was there evidence of "organic disease" in the generally accepted sense of that term. In six, there was present focal infection to a degree sufficient to account for malnutrition. Three patients showed definite anemia of the secondary type. Twenty-two patients showed definite evidences of deranged metabolism, the type of derangement indicating endocrine malfunction. An analysis of these evidences of deranged metabolism shows that they were due to thyroid failure in eight cases, to pituitary dysfunction in seven cases, and to gonad failure in seven cases, the latter all females.

In this group of endocrine cases, the history disclosed certain important features. Eighty-two per cent of these patients complained of abnormal fatiguability, as contrasted with 22 per cent of the non-endocrine group who gave a similar history. This symptom of abnormal fatiguability always suggests improper functioning of the bodily machinery, and if physical examination discloses no "organic" disease to account for it, is indicative of metabolic derangement. Forty-one per cent of the endocrine cases complained of tinnitus, and

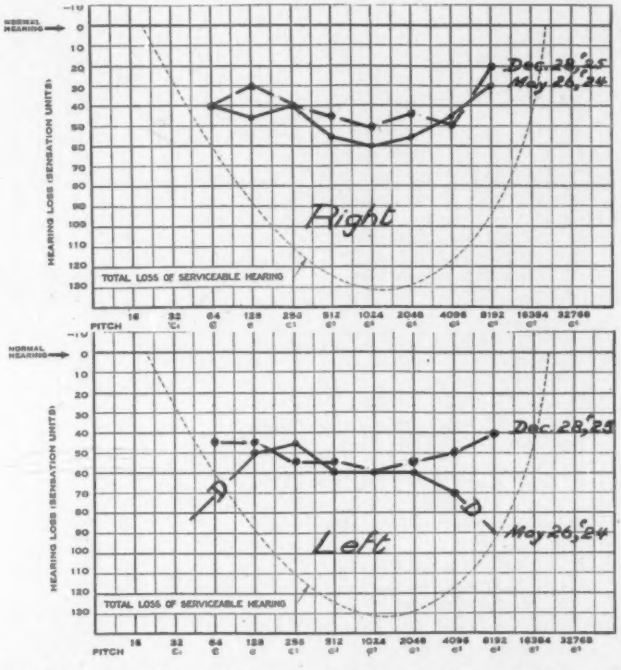
9 per cent of vertigo, while neither symptom was present in the nonendocrine group. Another significant bit of evidence obtained from the history was the age at which catamenia was established in the female patients. Twenty-one of the twenty-two patients with endocrine dysfunction were women. Of these, menstruation did not begin until the fifteenth year or later in twelve, and in four others was irregular. This failure of the catamenia to be normally established is another important indication of abnormal nutrition.



Case B-198. Female, 52 years. Diagnosis: Hypogonadism, surgical. Deafness, tinnitus and headache following surgical castration. Chart shows effect of 3 months' treatment with ovarian extract by mouth.

Physical examination of these patients was of importance mainly to disclose or eliminate organic disease. The diagnosis of endocrine disorder was not made in any case in which any other condition could be demonstrated. In this connection it should be noted that chronic infection of the nasal accessory sinuses was the undiagnosed condition most often encountered in the patients classed as nonendocrine —an indication that it is frequently overlooked when it is not causing local symptoms.

Besides eliminating organic disease, physical examination showed in the majority of cases definite evidences of malnutrition. Nine patients were more than 10 per cent underweight and four were more than 10 per cent overweight according to the Dreyer standards, a total of 59 per cent whose weight was distinctly abnormal. Blood pressure estimations were likewise indicative of malnutrition, as 90 per cent of the endocrine cases showed definite hypotension,



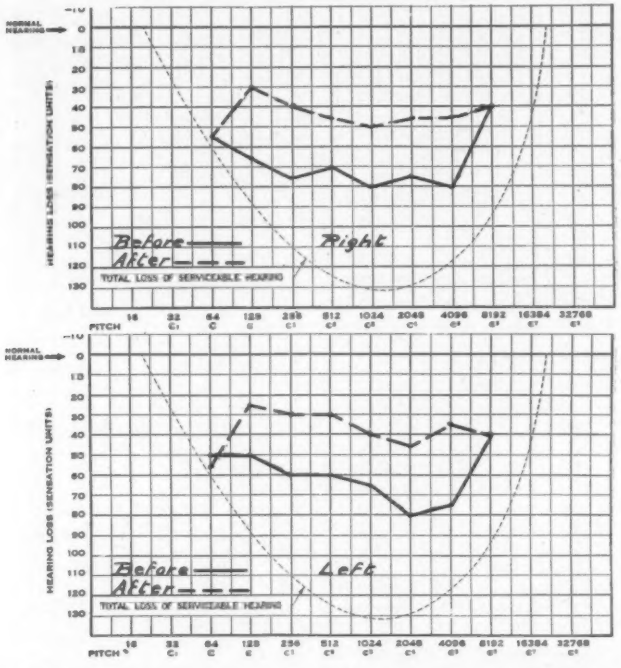
Case S-207. Female, 18 years. Progressive deafness, tinnitus, obesity, nervousness, 2 years' duration. Diagnosis: Pituitary dysfunction, anterior lobe underactive, posterior lobe not determined. Chart shows result of treatment with anterior lobe pituitary extract by mouth.

often as low as 95 m.m. systolic and 60 m.m. diastolic, while the nonendocrine cases uniformly showed normal or very slightly depressed readings. The laboratory findings are given in Table III.

Briefly, fourteen, or 63 per cent, showed basal metabolic rates below normal; seventeen, or 77 per cent, showed subnormal alveolar carbon dioxide tension; twelve, or 54 per cent, showed abnormally high amounts of undetermined nitrogen in the urine, and five, or

22 per cent, showed an increase in one or another of the blood nitrogen constituents. The Wassermann reaction was negative in every case.

Based upon the history, physical examination and laboratory findings, the cases showing evidence of nutritional disorder due to endocrine malfunction were grouped as follows (see Table I): Eight cases were diagnosed as due to thyroid hypofunction, seven cases

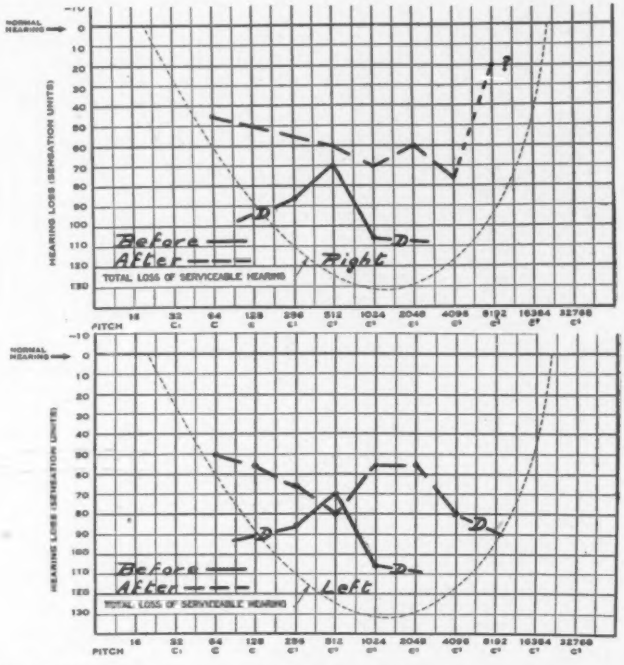


Case B-238. Female, 29 years. Progressive deafness and vertigo, 8 years' duration. Diagnosis: Pituitary dysfunction, anterior lobe underactive, posterior lobe overactive. Chart shows results of treatment with anterior lobe pituitary extract by mouth.

as pituitary dysfunction with the anterior lobe under, and the posterior lobe overactive, and seven cases, all females, as gonad hypofunction. The fact that the deafness was associated practically equally with derangements of three different endocrine functions is evidence that there is no direct relation between any internal secretion and the auditory mechanism. The influence of the endocrine system upon hearing depends upon the fact that the former regu-

lates metabolism, and that the latter is to some extent dependent, for its efficiency, upon the level of metabolic function in the body. Only by disturbing nutrition can the endocrines affect auditory efficiency.

To further test the relation between endocrine disorders and progressive deafness, nineteen of the patients were given treatment with endocrine extracts. The effect of this treatment upon the

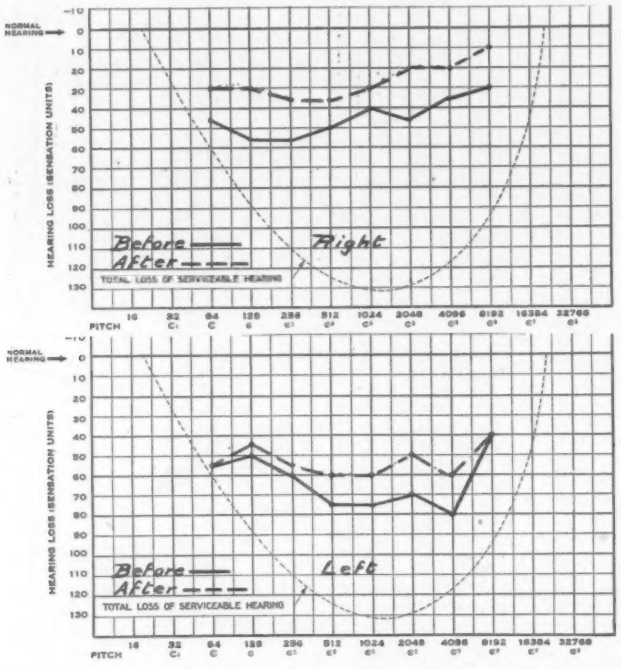


Case B-130. Male, 53 years. Progressive deafness with tinnitus, 8 years' duration. Diagnosis: Pituitary dysfunction, anterior lobe underactive, posterior lobe overactive. Chart shows results of treatment with anterior lobe pituitary extract.

hearing and metabolism was determined from time to time thereafter. All the cases reported in this series have been under treatment at least six months; the longest period of observation has been eighteen months.

Reference to Charts 1-8 shows the results of treatment, and comparative audiogram records in several cases. To summarize, fourteen patients, or 73 per cent of the treated series, have shown

some improvement; five, or 27 per cent, have shown none. The improvement is classified as marked in four patients. They have become, and remained, free from tinnitus for a period of six months or more, and are free from any conscious handicap due to deafness. Their metabolism has also become practically normal during the same period. Five patients have shown definite improvement, as evidenced by successive tests with the audiometer,



Case S-407. Male, 40 years. Progressive deafness, tinnitus, 10 years duration. Diagnosis: Thyroid failure. Chart shows result of treatment with thyroid extract.

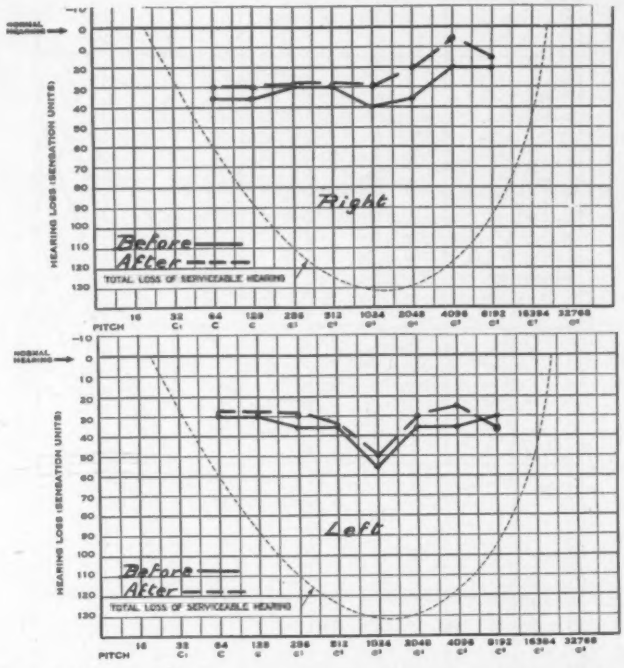
but are still somewhat handicapped by deafness. Tinnitus has been diminished but not eliminated.

Five patients have shown slight improvement only, *i. e.*, the audiogram charts show improvement for certain tones, but the patients are not practically benefited.

Five patients have shown no response to treatment, either as regards deafness or metabolic derangement.

In the nonendocrine group, two patients in whom the deafness was associated with anemia have shown definite improvement in hearing as the blood picture has approached normal.

An analysis of the unimproved cases suggests certain factors possibly operative in determining the results of treatment. One of these is the chronicity of the deafness. The duration of impairment in the unimproved cases averaged 9.8 years. A process of such long

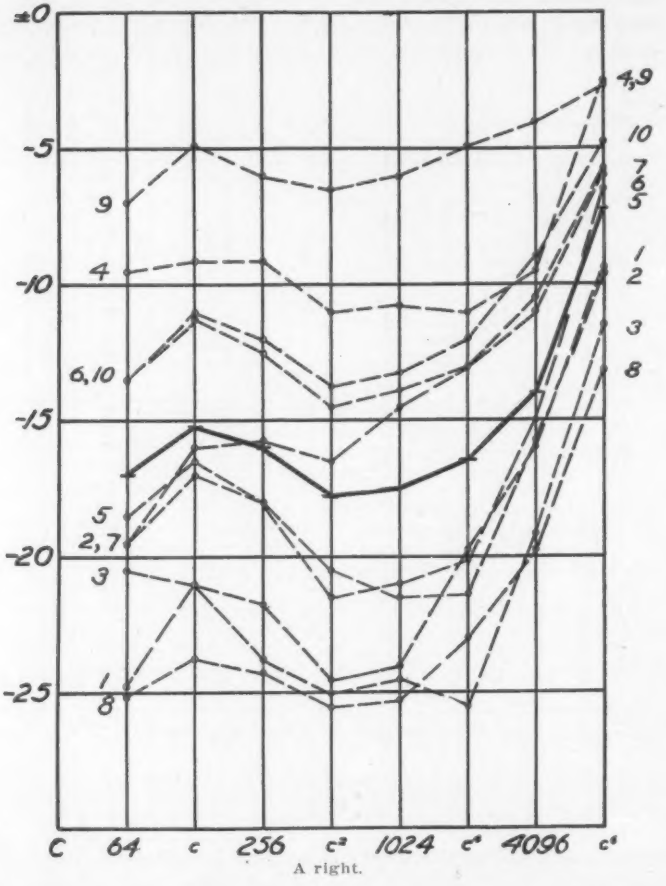


Case S-448. Female, 38 years. Progressive deafness with tinnitus, 3 years' duration. Diagnosis: Pituitary failure, bilobar. Chart shows results of treatment with extract of whole pituitary gland by mouth.

duration would tend to become fixed, and an improvement in hearing was not expected. That this is not the sole factor, however, is shown by the fact that five of the improved cases had been deaf for ten years or over when treatment was undertaken.

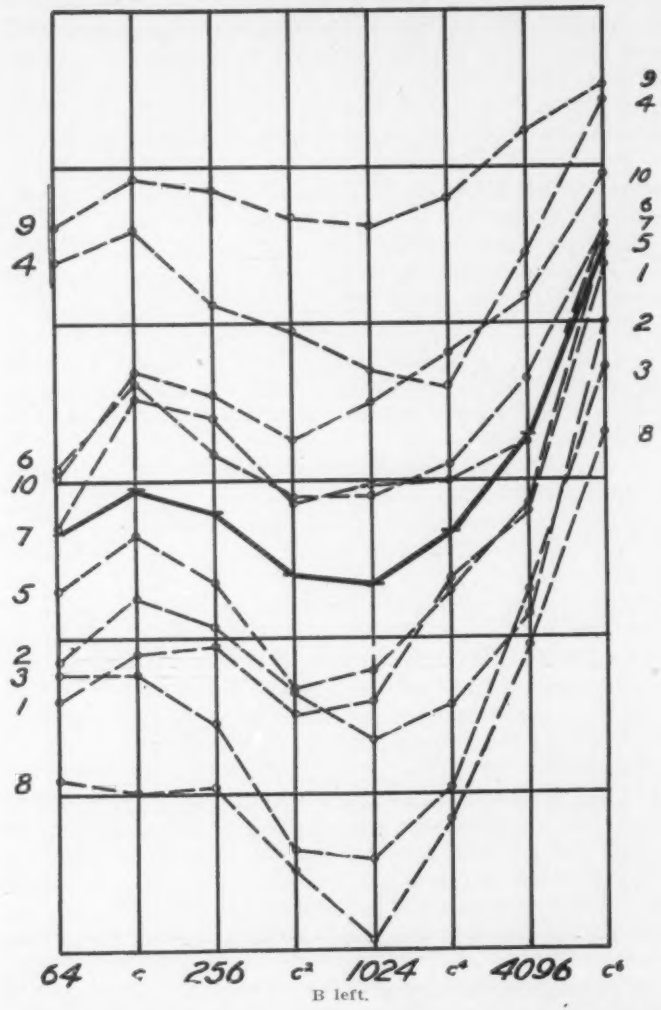
The other definite factor which influenced the result of treatment was the endocrine function which was disturbed. (See Table IV). The cases of thyroid or pituitary failure include eight of the

nine classed as markedly or definitely improved, and four of the ten in which improvement was slight or absent. In contrast the cases of ovarian failure embraced only one of the nine in which improvement was marked or definite, and six of the ten in which it was slight or



A right.

lacking. This difference in results is in all probability not due to the specific effect of ovarian failure upon metabolism, but to the lack of an efficient preparation of ovarian extract with which the condition can be treated. The failure of various preparations of ovarian substance to show any effect in cases of ovarian failure is generally



B left.

Charts A and B show the average hearing of 500 individuals without disease and with supposedly normal hearing. The heavy black line indicates the average for the entire group. The broken lines indicate the average for various subgroups, as follows: Group 9, college athletes; Group 4, trained telephone operators; Group 10, music students; Group 6, private school children; Group 1, medical students; Group 2, trained nurses; Group 3, untrained telephone operators; Group 8, hospital school children.

agreed upon, and is the rule in a large series of such cases which we have studied. The fact that medication had no demonstrable effect upon metabolism or hearing is a further bit of evidence concerning the interrelation of aural function and nutritional level.

Of all cases treated the thyroid failure group responded most rapidly to medication. Improvement in the patients with pituitary failure was less rapid, and occurred only when generous amounts of the extract were administered. All medication was by mouth. By that route, the effective dose of pituitary extract was between 16 and 30 grains daily. Until endocrine products are measured in physiological units rather than weight the dosage must remain indefinite and unsatisfactory.

CONCLUSIONS.

1. Thirty-one cases of progressive noninfectious deafness (otosclerosis) in which the metabolism studies were made are reported. Of these 22, or 70.9 per cent, showed definite evidence of metabolic disturbances.

2. The nature of these disturbances indicates that their origin lies in endocrine malfunctions.

3. The unfavorable effect of pregnancy upon deafness of this type may be explained upon the basis of its influence on metabolism *via* the endocrine apparatus.

4. The auditory apparatus is peculiarly sensitive to systemic conditions, either because of its developmental characteristics, or in certain individuals, of an inherited lowered resistance.

5. Treatment of the metabolic derangement associated with the deafness by administration of an extract of the endocrine gland at fault resulted in improvement of hearing in 14 out of 19 cases—73.6 per cent.

6. This paper is intended as a preliminary report. Convincing evidence can be adduced only after the study of a much larger series of cases. Such a study is now in progress at the Evans Memorial.

I wish to thank Dr. Drury, who conducted the otological examinations, for his co-operation; Dr. Hunt for his help in charting the findings; and the Bell Telephone Company for the use of the audiometer.

520 Commonwealth Avenue.

THE MAINTENANCE OF ATTITUDE AND ITS RELATION TO THE VESTIBULAR MECHANISM.*

DR. J. GORDON WILSON, Chicago.

The work that has been presented, from the Universities of Utrecht and McGill (THE LARYNGOSCOPE, Oct., 1926, pp. 701-728), based on beautiful and accurate experimental work, establishes beyond doubt the tonus labyrinthine theory of Ewald, that the labyrinth of the ear exerts an influence on certain muscles of the body. This fact stands secure though some of Ewald's hypothetical deductions have had to be discarded. I am indebted to Professor Magnus and also to Professor Tait and Dr. McNally for sending me copies of recent publications which enables me better to concentrate on what I have to say.

I. THE PAPERS OF PROFESSOR TAIT AND OF PROFESSOR MC NALLY.

The careful way in which these experiments were conducted and every conceivable error avoided, calls for nothing but praise. One hesitates to question any of these conclusions supported as they are by mathematical and physical data. Yet this thought keeps recurring to me while reading the recent articles of Professor Tait (Tait and McNally, Rotation of Acceleration Experiment, Mainly on Frogs. *Am. Jour. of Physiol.*, Vol. 75, 1925, pp. 140-154, pp. 155-179) are we stressing angular acceleration in our explanation of the attitudes assumed? I raise this question in connection with the explanation given of the attitudes assumed by the frog rotated tangentially with the value of the acceleration rising steadily in magnitude. The frog abruptly assumes certain postures and assumes them in shifts. The explanation given of this shift is "that the frog has a certain range of selection in the matter of posture and does not squat or adjust itself to lateral thrust in the same way." (Tait and McNally, loc. cit., p. 150.)

It is agreed that the stimulus for the semicircular canals is angular acceleration. But if we conclude our investigation of change of attitude with the mechanical factor we lose an important part of its causation and a restricted view of the process involved.

If one is seated in a chair so that the back and body are fixed and the chair slowly rotated with a constant, very slow acceleration starting at zero the resulting sensations are: (Dodge: Threshold of Rotation. *Jour. of Experimental Psychology*, 1923, Vol. VI, 2, p. 126).

*This paper was prepared in conjunction with the Symposium by Drs. Magnus and Tait at the joint meeting of the American Otological Society and the American Laryngological, Rhinological and Otological Society, Montreal, June 2, 1926.

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a. Consciousness of motion with no knowledge of direction, alternating with periods when no motion is felt.

b. Passing into a period when correct rotation may be sensed or where a reversal effect may be sensed.

c. Gradually as the speed increases the error of misjudgment diminishes till one gets a fairly accurate knowledge of the direction of rotation.

During these periods the mechanical factor is unaltered except in regard to increasing speed. I cannot see how a constantly directed speed *per se* can give such varying impressions if we regard these impressions solely as the result of the mechanical factor. Something occurs when this physical energy is translated into nerve energy. Let us remember that the degrees of contraction of a muscle are not as numerous as the degrees of strength of the exciting stimulus but take place in a series of steps. (Bayliss: Principles of General Physiology, 1924, p. 383). There is needed, as the physiologist says, an increment in the intensity of stimulation before the effective result comes.

Apply this to the frog rotating with the acceleration rising steadily in magnitude. The frog abruptly assumes certain postures and assumes them in "abrupt shifts." In other words, each shift requires a definite increment of energy before the shift is made, just as one requires a definite increment of frequency before recognizing the difference between pitches or of amplitude between two sensation units. I prefer this point of view since it places the emphasis where it belongs on the nerve mechanism and less on the mechanical action of acceleration.

In their first summary Tait and McNally (loc. cit., p. 153), state that "to change of simple linear acceleration in the line of gravity, as in an elevator, there is little or no reaction on the part of a normally poised animal; to change of linear acceleration in a direction other than gravity the animal reacts by some compensatory movement." This appears to me open to objection. There is certainly compensatory reaction, though there is no resultant compensatory position observable, because the resultant is the normal attitude. Were we to accept this summary as it stands we would be led astray in the understanding of certain pathological cases.

II. THE RESULTS OF REFLEXES FROM OTHER SENSE ORGANS ON THE LABYRINTH—CO-OPERATION AND INTERFERENCE.

Dr. Magnus, in his interesting paper, has given us the results of the brilliant experimental investigations which he and his collaborators have carried on at Utrecht for many years and which in its main outline has been brought to an approximate conclusion. They have analyzed the mechanism involved in the various postures which the

body can assume, located the place of origin of the stimuli and pointed out the adjustment of the reflexes which result in adequate muscle action. As a result much of the confusion and ambiguity which surrounds the problems of equilibrium is being cleared away. Attentions is focussed on the anatomical and neurological sides of the problem to which as clinicians we must ultimately refer our difficulties.

There are two thoughts which should ever be present in discussing the intricate subject which Professor Magnus has so briefly and so clearly presented to us:

1. That the central nervous system in man is a unit into which fit several more or less independent units with special functions of which one, equilibrium, has an important part of its mechanism located in the labyrinth.

2. So far as the labyrinth is concerned when we consider it at different stages of vertebrate development the anatomical mechanism used has not a fixed localization. There is apparent a flexibility in the mechanism with fixation of function.

The central nervous system controls the movements and the attitudes of the body not only in relation to gravity but in relation to movements in the horizontal plane. It is the central nervous system which regulates and distributes the quantity of impulse allowed to pass out to the appropriate muscles. To do so adequately it must have information in regard to the attitudes of the body or movements of the body which are being carried out. It is to assist in supplying such information that is the chief function of the labyrinth. Involved in the complete reactions necessary for equilibrium we have therefore three factors:

1. The adequate afferent stimuli—from various sources—which enter into its inception.

2. The co-ordination of these stimuli—the summing up and adjustment of the stimuli, the avoidance of conflicts.

3. The adequate muscular reactions—the efferent pathway through which they are made effective.

It is with the first of these we are here chiefly concerned—biologically adequate stimuli.

BIOLOGICAL ADEQUATE STIMULI.

When one analyzes the afferent impulses which set a mechanism going one does not require much physiological acumen to be impressed with the number of responses in which afferent impulses from more than one source can be shown to participate. One may well doubt whether a response of the skeletal muscles in the higher vertebrates ever involves afferent impulses from one source only. Frequently it is the accessory afferent impulses, the impulses arising

from sensory fields other than the limited one to which a given stimulus is applied, which makes the reflex response adequate for the particular movement required by the animal. It is true that a reflex response may occur in the absence of one or more of the afferent impulses normally entering into its control. In the intact animal and especially so in the higher vertebrates it is extremely doubtful whether a reflex unaffected by accessory reflexes ever arises. Take for instance, the maintenance of equilibrium. There is the liberation of one response by one preceding it, and there are the considerable number of afferent impulses which are involved in the reflex responses which result in the maintenance of equilibrium. The play and interplay of these afferent forces are bewildering in their complexity, and will remain so unless we recognize the underlying essentials. It is the admirable work done in Utrecht that has clearly established the sources from which these impulses arise and the way in which they work and co-operate. These investigations show that to make labyrinthine reflexes adequate accessory impulses nonlabyrinthine play an important part.

The study of the specific reflex responses to stimulation of a given afferent nerve and of the modification of any given response when any particular component of the afferent group is lacking, is of importance not only from the point of view of the physiologist, but also from the point of view of the clinician. As clinicians, one aid in our diagnosis is the study of the modification of typical motor responses which occurs when any particular afferent channel is blocked, for instance, the alteration of the normal reaction to caloric stimulation. Parenthetically it may be noted that here we are sadly lacking in knowledge. What is urgently needed is more precise statement of facts observed and more complete knowledge of pathways, on the lines of the excellent work of Jones and Spiller which has recently been published. (Jones and Spiller: Central Tracts of the Nervus Vestibularis. *Brain*, Sept., 1925.)

One can get an objective demonstration of the fact that afferent impulses from at least two different sources are involved in the activity of the muscles which maintain the position of the head. If one otic labyrinth of a cat is extirpated, there is torsion of the head, the occiput being turned toward the injured side and the nose toward the sound side. If, after an interval varying from one hour to several months the dorsal roots of the cervical nerves of the opposite side are divided, the torsion of the head disappears. (Prince: *Amer. Jour. of Physiology*, 1916.)

The relationships of the various afferent impulses from different sources are not always obvious. In man especially more or less of a subjective element may enter into the demonstration. There is also

a possibility that when one of these afferent channels is blocked by accident or disease, certain of the other channels may, by an increase in the quantity of energy which passes over them, compensate, in part at least, for the loss of the impulse which formerly came in over the damaged pathway. In saying this I am not forgetful of the all or nothing theory of Adrian.

POSITIONAL REFLEXES.

In the study of positional reflexes in vertebrates we note that while there is a continuity of function this is modified in anatomical location by a progressive development. It is to this aspect of the problem that I would for a moment refer, since it appears to me that it has a bearing on the application of the work here presented to problems which we see in man. In the study of positional reflexes from the labyrinth one must clearly understand what is meant by that term. Positional reflexes are those unconscious compensatory positions which persist after movement and which are retained so long as the head remains in a particular position in space. They are to be distinguished from reactions incidental to movements which are transitory, for instance, in the eyes produced by angular acceleration or by the caloric reactions. They are also distinguished in the source of origin of the stimuli, the positional, chiefly at any rate, in the otolith organs, the caloric in the semicircular canals.

Positional reflexes from the labyrinth and tactile organs are to be observed in vertebrates from fish to mammals. Considering those arising from the labyrinth we note from the evidence at hand there is a shift in the location of origin of the reflexes at different levels of the vertebrate phylum and a modification in the co-ordinating mechanism involved. In such reflex animals as rabbits and guinea pigs, in which these reflexes can be well observed, there appears little doubt from the work of Magnus that they originate in the otolith organ. But Maxwell clearly showed that in fish the positional reflex or, as he called it, sustained forced position could come from the ampulla. Here then we note a shift in anatomical location, so far as origin of this reflex is concerned.

Eye positional reflexes in the rabbit and guinea pig according to the evidence presented here today come from the otolith of the saccule. Though of great complexity in their ultimate summation, on analysis De Kleyn found that the tonic reflexes from the labyrinth, which modify the eye position in the orbit, result only in a vertical or rotary movement. No sidewise motion of the eyes, that is, in the direction of the palpebral fissure, was seen. The source of these reflex movements in the rabbit was the saccule. But in fish where the saccule is well developed Parker and Maxwell have clearly shown that the loss of this saccular otolith "does not alter or weaken any of

the compensatory movements, it does not disturb equilibrium or the righting reflex, nor is the muscle tonus affected in any way." Here then is a second functional change in location.

Again, tonic labyrinthine reflexes acting on the muscles are modified by the position of the eyes in the head, lateral or frontal. In the rabbit with laterally placed eyes where the images do not fall on corresponding points of the retinae these labyrinthine reactions are marked. In the monkey and man where the images fall on corresponding points of the retinae they are "small and subsidiary" to ocular positional reflexes. Here there is a third functional change during phylogenetic development.

A fourth functional modification is apparent when we consider what may be called the ocular component in such spinal animals as the guinea pig and rabbit. In these animals the co-ordinating mechanism adjusting the tactile, the neck and the labyrinthine reflexes, the positional and righting reflexes, has been shown to be in the medulla, pons and mesencephalon. But with the development of the cerebrum in the higher mammals the primitive co-ordinating mechanism located there has I believe been modified by a shifting forward of co-ordinating areas. Thus in the eye positional reflexes there is a difference in mammals according to their position in the phylogenetic scale. There is introduced an ocular component, so manifest in monkey and man, dependent on the activity of a high cerebral factor which ultimately becomes the controlling factor in their positional reflexes. When we talk of the ocular movement as a factor in equilibrium we ought to have a clear idea of what we mean. The ocular component as I understand it, comprises the retina and the ocular muscles. The retina is vision and position is muscular, and vision and muscle sense are not interchangeable. Each nerve of special sense, no matter how excited, gives rise to its own peculiar sensation. The retina can only act through associating with positional muscles and through an integrating mechanism, and into this co-ordinating mechanism labyrinthine and neck reflexes go. In relation to this we get a modification in function by the introduction of the quick component of nystagmus—not present in lower vertebrates but clearly present in the higher mammals. As a result of the experimental work of Dr. Pike and myself on dogs and monkeys and as the result of our clinical observations we believe that the quick component involves a different pathway and higher cerebral association than can be explained from the red nucleus or any nucleus caudal to it by Dr. Magnus and his co-workers.

THE RIGHTING REFLEX.

By no means the least interesting part of Dr. Magnus' work has been his critical examination and his explanation of the righting

reflex. This reflex has been clearly defined by de Kleijn (de Kleijn, A.: Experimental Physiology of the Labyrinth. *Jour. of Laryng. and Otol.*, 1923, p. 655), as follows: "If a normal guinea pig is held in the air by its loins and the trunk is brought into different positions in space, the head always tends to remain in the normal position and does not conform to the different positions of the trunk. Breuer showed that this phenomenon must be ascribed to labyrinthine reflexes, because after bilateral labyrinth extirpation it disappears. These labyrinthine "righting" reflexes acting on the head can be studied in normal guinea pigs and rabbits; in all other animals the cerebral hemispheres must first be removed, but the mid-brain (Magnus), especially the nucleus ruber, must be left quite intact because the centres of these reflexes are situated there. (Rademaker.)"

Professor Magnus has shown that in a rabbit lying on its side, and deprived of influences from the cerebral hemispheres, the following reactions occur:

1. The stimulation arising from the labyrinth causes an asymmetrical distribution of tonus on the neck muscles which compels the head to assume a position of symmetry with relation to gravity, that is, a turning of the head into the normal position.

2. This turning of the head into the normal position results in an asymmetrical relation of the head to the trunk. As a result afferent tonic stimuli from the neck (joints and muscles) act on the trunk to compel the body to assume normal posture or symmetry. So the body and limbs follow the head.

3. This action is aided by tactile stimuli from the side on which the body lies.

While the normal rabbit shows no visual righting reflexes and behaves as does the mid-brain rabbit, dogs and cats show the influence of the cerebral cortex in the activity of visual righting reflexes. Magnus found that the normal dog with labyrinth intact depends on this source for its orientation in space and only falls back on the eyes when the labyrinth is unable to function. Several years ago we found (Wilson and Pike: The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System. Royal Society, London. Phil. Trans. 18, B. V. 203, p. 143) that a dog deprived of both labyrinths, put into a pond, struggled about in a helpless way with the body in all kinds of attitudes and that it had to be removed or it would have drowned. But if we repeated this the animal acquired a facility of movement which precluded drowning, a facility associated with vision. Are we here looking at the development of an associated path which an emergency is calling into play? In monkey and in man compensatory eye positions from the labyrinth and neck obey fundamentally the

same laws as in the rabbit. However, whereas in the latter animal these compensatory eye positions play a very great rôle, in apes and man this rôle is very greatly diminished.

I went the other evening into the children's ward and was interested in the various attitudes assumed by the sleepers. One child was lying not ungracefully half on his side,, half on his abdomen, with a twist at the neck. What was happening to his mesencephalic righting reflexes—were they dulled by sleep? Such an explanation appears inadequate when I recall the reaction of the labyrinthine reflex even in semi-comatose individuals. Is there in sleep some intermediate state exerting a control over these righting reflexes? If so, where is it—above the mesencephalon?

In the lower vertebrates and mammals there is a great and lasting loss of muscle tone after labyrinthine destruction, in monkey and man a good muscle tone persists and its loss is hard to demonstrate. Yet I believe that the primitive vestibular mechanism persists in all vertebrates but in higher vertebrates there is an ever-increasing association with centres above the mesencephalon. This does not mean a retrogression of the vestibular system in man but a higher degree of adaptability on the part of a more complex motor system.

One word in regard to the inhibitory factor. Experimentally by decerebration we cut off the so-called inhibitory influence and then consider *only* the effect of its absence on a centre which very probably because of this cutoff has augmentation of afferent force to it from other sources. But what was this "factor" doing before it was cut off? Inhibition is a positive and not a negative quantity. Suppose an inhibitory centre *A*, located according to our hypothesis above the mesencephalon, is controlling the red nucleus. To do so adequately centre *A* must get information from the periphery concerning equilibrium and if so such peripheral influences must pass up higher than the brain stem and the red nucleus.

I do not say that the red nucleus and the cells which extend from these to the medulla may not act in part as Dr. Magnus has outlined, all I plead for is the consideration of the possibility and likelihood that some part of the function of these areas has been transferred in man to other and higher areas, or at any rate that profound influences above the mid-brain are to be seriously considered. I believe that we should not forget the modifications in phylogeny which have already occurred, and that we should not be too dogmatic in transferring experimental data from rabbits to man. Rather let us corelate the experimental facts so clearly stated by Professor Magnus with observed pathological results seen in our clinical cases, for along this road lies progress.

104 S. Michigan Avenue.

THE INCIDENCE OF OTITIS IN 15,000 ACUTE EXANTHEMATA, WITH A REPORT OF THE METHOD OF HANDLING SCARLET FEVER OTITIS MASTOIDITIS AT THE PHILADELPHIA HOSPITAL FOR CONTAGIOUS DISEASES.*

DR. FRANCIS V. GOWEN, Philadelphia.

From a perusal of the literature concerning the ear complications of the acute exanthemata one gathers very little definite information as to their incidence. It seems that there has not been a very successful effort to determine the per cent of ear complications in these diseases. Much, of course, has been written on otitis and mastoiditis and their treatment.

At the Philadelphia Hospital for Contagious Diseases, we have one thousand beds. This is the biggest plant on this planet devoted to this kind of work. True, other cities, on account of larger populations, handle more contagious diseases, but they have the work divided among a number of hospitals, so, on account of our centralization, we are better fitted to study ear complications from big figures.

As mentioned in an earlier paper on ear complications in 10,000 scarlet fever patients at the Philadelphia Hospital for Contagious Diseases, we found that out of a total of 10,086 scarlet fever patients there were 1,063 who developed otitis, and of these, 210 a definite surgical mastoiditis. The percentage of noted cases is 10 per cent otitis, and 2 per cent mastoiditis.

Now, we are able to give additional information—

of 839 measles patients, 93 developed otitis and of these, 6 had surgical mastoiditis; about 10 per cent otitis, and .8 per cent mastoiditis.

of 172 pertussis patients, 9 had otitis and none mastoiditis; about 4 per cent otitis.

of 100 chickenpox patients, none had otitis and none had mastoiditis;

of 188 smallpox patients, 4 had otitis and none mastoiditis. All the 4 were children. The total number of children having smallpox was 45.

When the original disease became complicated by another acute exanthem, the percentage of otitis became greatly increased, *viz.*

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scarlet fever was complicated by measles in 74 patients, 29 developed otitis, and 4 surgical mastoiditis, or about 40 per cent otitis and 5 per cent mastoiditis;

scarlet fever was complicated by both measles and chickenpox four times, 3 of them developed otitis and 1 surgical mastoiditis, or 75 per cent otitis and 25 per cent mastoiditis.

scarlet fever, measles and diphtheria coexisted eight times, 4 developed otitis and 1 surgical mastoiditis, or 50 per cent otitis and 12 per cent mostoiditis.

We had one patient with scarlet fever, measles and German measles without any ear complication.

We had one patient with scarlet fever and German measles who developed otitis only; and we had a patient with scarlet fever and chickenpox without any ear complication.

To demonstrate the value of myringotomy, one interne skilled in the art of handling ear infections, over a period of three months had charge of a ward of 96 patients, including scarlet fever, diphtheria, and various combinations of the acute exanthemata, and in that time he had only three surgical mastoids, while, in the period of ten days after this transfer, four mastoids developed.

With regard to discharge from the ear, we wish to repeat from our former paper, that the drum ruptures easily and readily in childen, and the period of pain is short or not present at all. We wish to emphasize here the fact that in more than two-thirds of bulging drums in scarlet fever otitis in children under our care, where one would expect pain, no complaint was made.

ROUTINE HANDLING OF EAR INFECTIONS AT THE P. H. C. D.

All patients on admission have their eardrums inspected. If during the course of the disease, a fever without obvious cause develops, the eardrums are again inspected. Besides frequent syringing and wicking, bulging or pulsating drums are incised by myringotomy and not by stab paracentesis. Myringotomy is repeated as often as indicated. Shrinking solution is used to aerate the nose and keep the Eustachian tube open. Another procedure which we have employed with success and never any untoward results, is suction applied daily to the Eustachian tube by means of a Eustachian catheter and pump. By this method antiseptic solutions can be placed in the auditory canal and sucked through a perforated drum into the tympanum and out the Eustachian tube. Adenoids are removed routinely as soon as this procedure will not adversely influence the course of the initial disease, which is about the third week. The result of this procedure is generally striking as we often find a dry ear within two or three

weeks. However, we have seen a complicating otitis in the other ear following adenoidectomy, and we have seen surgical mastoiditis subsequent to extirpation of the adenoids. The tonsils are not removed routinely. However, none are permitted to remain which show removal indications.

With regard to the vaunted rapid destruction of the eardrum in scarlet fever, we at the P. H. C. D. do not see it; quite the opposite, in fact, exists there. We do see all forms of destruction of the drum and tympanic contents when the discharge has persisted over a long period of time, like two or three months, but as for the drum being entirely destroyed or even partly destroyed in a few days or a couple of weeks, we do not find it. We are able to say this after having looked into hundreds of scarlet fever ears and not finding it in any case. The usual thing is for the nurse or interne to call us to an otitis patient and find the straw-colored fluid still in the canal, wax not present, it having been melted away by the local heat, often detritus is present. Upon removal of the occluding substances, the drum is found blood red in color, landmarks are gone, extreme bulging is not necessarily present, pulsation is often present, probably caused by the hyperemia. Earlier in our career we would do a stab paracentesis and return the next day or so and still find a pulsating drum and then we would repeat the procedure and repeat it several times; whereas now with the wide open myringotomy with a flap, very seldom do we have to reopen the drum and fewer mastoid operations result.

In regard to the rapid destruction of bone in scarlatinal mastoiditis, we are inclined to agree with Strachan that it occurs in neglected cases; we believe with Salomunsen and Marshall that if the mastoid is opened as soon as it is tender and infiltrated widespread destruction of bone will not be found.

The operative technique for the treatment of mastoiditis is as follows: The drum membrane is incised if indicated. The skin is painted with iodin. The usual skin incision is made as close to the auricle as feasible to treat the subsequent wound. The soft parts are retracted and the bone removed in layers from below up with hammer and gouge until access can be had to the antrum, which is entered with a curette. All diseased bone is removed. All the mastoid cells are removed. Passage between the antrum and the middle ear is assured by forcing salt solution in the antrum and out the ear canal. A cigarette drain to the antrum is inserted and the wound is closed to one-half inch of its lowest extremity. The adenoids are then removed. The dressing is first changed at the end of 12 hours,

the drain removed, the wound irrigated, and another drain consisting of a piece of sheet rubber inserted. Thereafter the wound is dressed daily by ourselves until about the fifth day, when the stitches are removed. If the drain is not then removed, it is removed in a day or two. The wound generally heals in two weeks, often in eight to ten days.

RESULTS OF MASTOID OPERATIONS.

These operations were done in the latter six months of the years 1922, 1923, 1924, and part of the latter six months of 1925.

The total number of scarlet fever patients handled was 3,028. Of these, 278 developed otitis, and of these 23 were operated upon for mastoiditis.

The total number of consecutive mastoid operations were 23; cures, 21; sinus thrombosis, not any; facial palsies, 0; brain abscess, 0; reoperations, 0; deaths, 2. The total number of unoperated surgical mastoiditis were 0.

One death was due to pneumonia fourteen days after the operation. This child also had the rickets. The other was due to streptococcic meningitis, which will be described.

In considering the mortality rate your attention is invited to the fact that this operative work was done on poor risks. Practically all of them were children from poor families. Most of them were undernourished. All of them were devitalized by one serious disease and endangered by nephritis and other grave complications; and many of them had mastoiditis for some time before being sent into the hospital. At that, our mortality rate for this especially serious type of mastoiditis is well within the limit of Mygind, whose mortality in one thousand mastoids was 10 per cent.

With the end in view of preserving hearing and especially of preserving life, we adopted the policy in all otitis cases of watchful waiting. As soon as mastoiditis occurred we operated, getting in early. When conditions were such as to render the operation more dangerous to the patient than conservative treatment, we waited and saw the patient daily and did efficient myringotomies, and kept the tube open with shrinking solution, as well as the rest of the routine handling as mentioned above until the ear healed or demanded intervention.

REPORT OF CASES.

We have two cases we would especially like to report:

Case 1: Streptococcic Meningitis Complicating Mastoiditis and Scarlet Fever.

L. G., age 6 years, admitted Nov. 3, 1924. Had measles, chickenpox, whooping cough, and had just recovered from the mumps. He

had a very rapid heart, and he had a soft systolic murmur. He had considerable postnasal obstruction and enlarged submerged cryptic tonsils.

Nov. 5, 1924, he developed rhinorrhea and adenitis.

Nov. 27, he developed otitis, right side.

Nov. 30, mastoiditis developed, right side. Fever 101,° auricle stands out, mastoid edematous and tender. Simple mastoid operation done, as described elsewhere in this paper. There was no macroscopic connection between the mastoid process or antrum and the posterior fossa. The lateral sinus was exposed, the dura was not. Patient's condition good after operation.

Dec. 1, dressings changed, drainage good.

Dec. 2, temperature 100,° condition good.

Dec. 3, temperature normal.

Dec. 4, incision infected, sutures sloughing. All sutures removed, general condition good.

Dec. 6, temperature normal.

Dec. 7, wound hemorrhage during the night, bandages changed.

Dec. 8-9-10, fever 102.°

Dec. 12, right ear discharging profusely. Left ear discharging slightly.

Dec. 13, temperature normal.

Dec. 14-15-16, fever 102 to 103.° Vomited twice on the 16th.

Dec. 18, myringotomy done on the left ear.

Dec. 19, temperature normal. This drop in temperature threw us off the track for a while. We thought the fever due to left otitis.

Dec. 21, discharge scanty on the right side, wound clean. Both ears discharging.

Dec. 21, patient drowsy, neck slightly rigid, respiration slow, pulse slower.

Dec. 21, lumbar puncture, cloudy fluid under pressure. Examination showed no organisms but many pus cells.

Dec. 21, 30 c.c. polyvalent antimeningococci serum given intraspinally.

Dec. 22, spinal puncture, 35 c.c. cloudy fluid withdrawn, 15 c.c. antimeningococcic serum given. Lumbar puncture repeated, 20 c.c. fluid being removed, 30 c.c. antimeningococcic serum given.

Dec. 23, lumbar puncture fluid flows very slowly, is very cloudy and some flakes. Patient not improved. All classical symptoms of meningitis present. Culture report shows pure growth of streptococcus hemolyticus.

Cysterna puncture done and 20 c.c. polyvalent antistreptococcic serum given.

Operation, chloroform anesthesia, mastoid incision enlarged. Skull exposed and trephined. Dura exposed and incised one-half inch, a free flow of cloudy fluid being obtained. Needles were inserted into cysterna magna and lumbar spinal canal and 20 c.c. Ringer's solution introduced into dural wound flowed out through cysterna needle. 20 c.c. Ringer's solution introduced into cysterna needle flowed out dural wound with flakes. Solution then introduced through the lumbar region and permitted to flow from needle in cysterna; when solution became fairly clear the lumbar needle was removed and 20 c.c. of polyvalent antistreptococcic serum was introduced into the cysterna. A probe inserted between the dura and the brain and pointed toward the base released quite a free flow of cloudy fluid with some flakes of pus. A very small subdural cigarette drain was put in and the skin wound closed. Patient returned to the ward in fairly good condition and recovered from the anesthesia readily.

Dec. 24, cranial wound draining freely a cloudy fluid, neck rigid, generalized convulsions, head drawn to the left, Kernig present. Cysterna punctured and 10 c.c. cloudy fluid withdrawn, 30 c.c. polyvalent antistreptococcic serum given in the cysterna. 20 c.c. of the polyvalent antistreptococcic serum given intravenously. 10 p. m., patient weaker, cysterna puncture done, fluid cloudy and 20 c.c. antistreptococcic serum given. Lumbar puncture, small amount of cloudy fluid obtained. 11:35 p. m., patient died, a day and a quarter after the operation.

Autopsy demonstrated a thick pyogenic membrane adherent to the internal surface of the cysterna magna with the other findings of a purulent meningitis. The thickness of the membrane and its adhesion makes us wonder whether any kind or any amount of irrigation would have dislodged it.

Case 2: W. N., age 2 years, admitted Dec. 2, 1924, with scarlet fever, pertussis, nephritis, and surgical mastoiditis, and developed nasal diphtheria while convalescing from his mastoid condition. The child was sent into the hospital on account of his mastoiditis. He was 2 years old, well nourished and robust. He was operated upon the same day he was admitted under chloroform anesthesia. Pus was found under pressure. Thirty minutes elapsed from the start of the anesthesia to the completion of the operation, including removal of adenoids. His ear became dry and his mastoid wound healed fifty days after his operation. In the meantime his other conditions cleared up. Coincidental with the healing of his ear he showed positive diptheria culture from his nose. Antitoxin quickly ended this and he was discharged, cured of all his troubles, shortly after.

In closing this paper I wish to acknowledge my indebtedness to Dr. Samuel Woody for his ready co-operation over a period of five years and through whose foresight such good records exist at the hospital. To Dr. P. A. McCarthy for his help in the neurosurgery of the meningitis case. To Dr. George M. Coates, who was so kind as to go over this paper for me. To my friend, Dr. George Morley Marshall, a distinguished professor of otology of the University of Pennsylvania, whose aid, advice and teaching have enabled me to do this work.

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1819 Spruce Street.

OTITIS EXTERNA HERPETICA AS A COMPLICATION OF THE PRESENT INFLUENZA EPIDEMIC.*

DR. M. REESE GUTTMAN, Chicago.

There is, at the present time a widespread outbreak of severe grip or influenza which is leaving in its wake an unusually large number of otitic complications. Contrary to the usual form of an acute otitis media, which has been seen in all of the past epidemics and which is caused by an extension of the rhinopharyngitis accompanying the influenzal attack up through the Eustachian tube, the lesion very frequently presenting itself now is a herpetic external otitis or bullous myringitis, the so-called herpes oticus of Hunt.

The occasional occurrence of herpes on the auricle or in the canal has long been known to occur, but Politzer,[1] in 1893, was the first to draw attention to its association with influenza, and only since Hunt's publications,[2] in 1906, have we had any definite conception of the true nature of the lesion and its basic pathology. He has shown conclusively that the lesion was due to and followed an

*From the Clinic of Drs. Joseph C. Beck and Harry L. Pollock and the Otolaryngologic Service of the North Chicago Hospital.
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inflammation of the geniculate ganglion and pointed out the similarity of herpes oticus and of other recognized forms of ganglionic or posterior poliomylitic inflammation as herpes zoster and its subtypes, herpes ophthalmicus, herpes pharyngicus and herpes branchialis. Furthermore, he has shown that the infrequent appearance of variations in the syndrome of otologia and herpes that exhibit in addition a facial palsy, profound deafness of the nerve type, or vertigo, nystagmus and vomiting were due to coincidental affection of the facial and auditory nerves as they lie in intimate anatomical relationship with the geniculate ganglion in the petrous portion of the temporal bone.

Ullman[3] has recently published an account of his experimental work that tends to establish a filterable virus as the causative factor in the disease. Under aseptic conditions the vesicular contents which were first cultured without success, were injected subdurally into guinea pigs with the subsequent production of a meningoencephalitis. Inoculation of the vesicular contents themselves or of the cerebrospinal fluid from one of the affected guinea pigs into the scarified cornea of a rabbit produced a violent conjunctivokeratitis progressing into ulceration. The virus could be successfully transferred from animal to animal, each time resulting in a similar meningoencephalitis when introduced into the subdural space or producing ulcerative conjunctivokeratitis when inoculated into the cornea, thus definitely establishing its specificity. It is not improbable that the specific cause of epidemic influenza or possibly one of its transmutated forms may be the offending factor in the production of herpes oticus.

The following description is based upon observation of thirty-one cases seen in the Clinic, in private practice, and in consultation. Individual case histories are purposely omitted, since they all conform more or less closely to the clinical picture to be presented.

The disease usually manifests itself during the declining stage of an acute influenza or just following an abortive attack. The patient first notices an excruciating pain in the affected ear, which may radiate over the side of the head, most frequently following the distribution of the great auricular nerve or the auriculotemporal branch of the trigeminal, that accompanying the former not infrequently simulating mastoiditis. The auditory acuity is not noticeably decreased except in those rare cases associated with involvement of the eighth nerve, which evidence profound deafness. The general phenomena are few, the temperature rarely rising to 99° or 100.° Many times a serous discharge occurs, but is small in amount.

This is due to a rupture of the herpetic vesicles and is never accompanied by that marked relief from pain that is usually associated with rupture of the drumhead in acute middle ear empyema. Inspection early in the disease will show a number of small crystal-clear vesicles, varying in size from that of a pinhead to that of a split pea. They are located most frequently upon the posterior canal wall or the posterior quadrants of the drum and are evidence of affection of the great auricular nerve. Less frequently, involvement of the auriculotemporal branch of the trigeminal will show blebs upon the anterior tympanic quadrants or the anterior canal wall. The tympanic membrane shows no evidence of inflammation except when it is the site of a blister, the inflammatory zone then being restricted to the vesicated area and the remaining portion showing no change whatsoever from the normal. The middle ear is rarely affected except secondarily, and then most frequently follows upon an ill-advised paracentesis of the drumhead. The blebs are of a pearl-white color, having a reddish areola surrounding the base. A network of blood vessels may be seen coursing over the vesicle if magnification is used. If it is punctured at this stage a clear serum will be obtained. Later the vesicular contents become clouded and opaque and not infrequently dark red, due to a hemorrhagic suffusion. More often the blister bursts spontaneously, leaving a small ulceration with a crust upon it. This soon becomes epithelialized and disappears. New crops only rarely follow on subsidence of the original eruption. Less often cases associated with facial palsy, extreme deafness or vertigo and nausea may be encountered, due to involvement of facial or auditory nerve. The affection runs its course from five to twelve days and is followed by uneventful recovery if not interfered with. The persistence of the neuralgic pain or of deafness or facial palsy are rare sequelae.

The diagnosis is established upon hearing a history of a preceding influenza or grippal attack and the otoscopic findings of normal drumhead and the vesicular eruption. Acute tympanomastoiditis (acute otitis media, mastoidism and mastoiditis) will evidence a markedly decreased auditory acuity associated with typical otoscopic findings of the tympanic membrane and positive radiologic phenomena. One must also consider the possibility of acute conditions of the external canal as furunculosis, vesicular eczema, otomycosis and also of an arthritis of the maxillotemporal joint.

The treatment is essentially one of the primary influenza with observance of a strict policy of noninterference. Topical applications of cocain (5 per cent), or of one of the newer cresol compounds, metacresylacetate (Cresatin), or of orthoform will afford

relief from the pain. The metacresylacetate is especially efficacious, resembling phenol in its local anesthetic properties, but without the destructive action of that drug. Puncture of the vesicle has neither caused diminution of the pain nor accelerated the disappearance of the lesion and is therefore contraindicated. Above all, the drumhead must not be incised as a secondary otitis media may result. Pyramidon or even codein may be required in addition to the topical applications to control pain. The resulting deafness or facial palsy requires no additional treatment as these disappear within a few weeks following the subsidence of the affection.

CONCLUSIONS.

1. Otitis externa herpetica is not an infrequent complication of influenza.

2. It is due to an infection of the geniculate ganglia with a filterable virus which infrequently also involves the facial and the auditory nerves.

3. The disease is self-limited and has no serious complications per se.

4. The pain accompanying the affection is most easily controlled by topical application of a local anesthetic.

5. Myringotomy is strictly contraindicated.

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2551 N. Clark Street.

ACUTE INFLAMMATIONS OF THE MIDDLE EAR.*

DR. MYRON METZENBAUM, Cleveland, O.

Reflex Hyperemia: The introduction of a snug-fitting speculum into the outer ear canal will cause an instant reflex hyperemia or injection of some of the blood vessels in the strip along the upper ear canal known as the stripe of Troelsch. This condition results in an increased amount of blood in the vessels which run from the superior canal wall to the malleus, then to the short process and then parallel to the posterior edge of the malleus, to the umbo. The malleus will then appear like a raised or elevated red strip on a pale drum membrane. In this case the middle ear is entirely normal or uninvolved.

Drum Indicates Acute Inflammations: The eardrum is such a delicate indicator of acute inflammations of the middle ear that in general all acute inflammatory processes of the middle ear can be recognized here, so that if the drum shows no signs of irritation then an acute inflammation of the middle ear can be ruled out. If, in addition, the patient has normal hearing, this speaks without reservation for a normal middle ear.

Subjective Symptoms: Besides the objective symptoms seen on the eardrum, there is also in every case of acute virulent otitis media, the subjective ear disturbances—pain, weakness, tinnitus, dullness of hearing, at times dizziness. These general symptoms are very important; their recognition under certain circumstances is very valuable for the diagnosis and treatment.

Earache: In acute otitis media, earache is the outstanding complaint and is always accompanied with tinnitus and some deafness.

Pain; Tinnitus: The pain is usually quite severe and with the tinnitus, which is always present, usually remains for a considerable period after the acute appearances of the inflamed eardrum has begun to subside or has even disappeared.

Dizziness: In the beginning of an acute severe otitis media accompanied by high fever, the patient often has subjective symptoms of dizziness. This symptom does not necessarily indicate an extension

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of the inflammatory process to the inner ear, but is usually due to the weakness of a very sick patient with a high fever. Such patients on sitting up or standing often sway and on walking they usually stagger, yet they are without the existence of any labyrinthine symptoms.

General Symptoms: In cases of acute otitis media the general symptoms are too seldom considered. The general conditions of a patient are very often a far better measure for determining the severity of the otitis media than the ear symptoms themselves. These, under certain conditions, may not indicate the true severity of the disease, while the poor general conditions may be the indication for an operation.

Fever: Every severe acute otitis media begins with a fever, which in the very beginning is often high, 39-40° C., and may be accompanied with chills and vomiting. In children, who do not always complain of their ears, and especially in infants, the physician naturally thinks first of the acute exanthemata diseases, acute intestinal disturbances or pneumonia. However, after a few days, a spontaneous purulent discharge may come from the inflamed ear, the fever subsides and there is established the real cause of the illness.

Pulmonary Involvement: On the other hand, children and infants who are suffering with an acute otitis media may have an accompanying pneumonia, which manifests so few symptoms that it is not recognized. Those children do not expectorate, cough but little and do not complain of chest pains. In such cases often the first to reveal the pulmonary disease is the fever chart, which remains continually high, even when the eardrum is perforated, discharging freely, and the ear pains have subsided. After five to seven days there is a sudden fall in the temperature to indicate that the child had a pulmonary involvement.

Double Otitis Media: In children and infants continued high fever may be due to an otitis media developing simultaneously in the other ear.

In the case of an acute otitis media which began with a high fever and where the fever persists after perforation of the eardrum with a free discharge and where there are no other complications, one is generally justified in considring that there is a progressive involvement of the middle ear and the neighboring bony and soft structures.

Middle Ear Infiltration: This suspicion will be confirmed if under our observation we see a wide ear canal grow narrower during the

course of an acute inflammation of the middle ear, especially if the swelling is in the depths of the ear canal in front of the eardrum and located in the superior posterior angle of the canal wall, this consistently proves the existence of a severe middle ear inflammation resulting from an inflammatory swelling of the upper ear canal wall following a periostitis emanating from the middle ear spaces. This inflammatory process may extend to the mastoid bone, resulting in a mastoiditis.

Infant's Ear Canal: Owing to the very narrow ear canals and the marked angulation of the eardrum in the infant an examination of the depths of the ear canal is often made only with the greatest difficulty.

Four Hour Temperature Chart suggests involvement of sigmoid sinus. The four hour temperature chart may give the first indication of the progression of the inflammatory process to the sigmoid sinus. High temperatures of short duration are most typical of sinus thrombosis, the patient has a chill with a sudden rise of high temperature, a profuse sweat followed by a remission of temperature. Such zigzag temperatures which repeat themselves often give the temperature chart a characteristic appearance.

Facial Paralysis: A further complication in an acute otitis media is a facial paralysis.

Cerebral Irritations: In children in the course of an acute otitis media very alarming cerebral symptoms may arise—stiffness of the neck, muscular spasms (somnolence), which suggest severe brain complications. However, as soon as the ear discharges spontaneously or through a paracentesis, these alarming symptoms may subside within a few hours. Evidently these symptoms are only the result of a transitory toxic irritation.

736 Rose Building.

THE LOCAL ADMINISTRATION OF BACTERIAL VACCINES IN THE TREATMENT OF SUBACUTE AND CHRONIC NASAL SINUS CONDITIONS.*

DR. HAROLD HAYS, New York.

In my presentation of this subject before the Laryngological Section of the American Medical Association in May, 1925, I gave a complete resumé of the work that we had been doing in the treatment of various nose, throat and ear conditions by the application of local vaccines. Since that time, we have applied vaccines locally in quite a number of cases and, with few exceptions, we have found the results eminently more satisfactory than when we used vaccines by the hypodermic method. Tonight I shall confine my remarks almost entirely to the treatment of sinus conditions by this method, although local vaccines can be used in any part of the body where direct application can be made.

It might be of interest to relate how I became interested in this method of treatment. During the summer of 1924, my attention was called to the work of Prof. Besredka, of the Pasteur Institute of Paris, who was able to prevent typhoid fever by the local administration of typhoid vaccine. His work proved that the ingestion of killed typhoid bacilli by mouth was harmless and that in many epidemics, in which people refused to be inoculated with a needle, they were perfectly willing to take a vaccine by mouth, with the result that epidemics were avoided as quickly as when the former method was used. The vaccine prepared for typhoid prevention was composed of killed, dessicated typhoid bacilli. Further researches proved that a broth suspension of killed bacteria could be made which could be applied directly to a surface and thus it came about that the vaccine was used in many cases of osteomyelitis and in various skin lesions, with excellent results.

A search of the literature reveals the fact that although vaccines had been applied locally as early as 1912, no appreciable data which stimulated research in this line began until after the published papers of Besredka. Since that time quite a little has been written upon the subject, with opinions for and against it. The method of

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preparation of the vaccine and the method of administration has varied. Much as we feel that there may be some variance from our opinion as to the efficacy of this form of treatment, nevertheless we shall present figures from our own clinical experience which have proved to us that, particularly in subacute and chronic nasal sinus conditions, the vaccine has a decided value and that the results compare more than favorably with other methods of treatment. We shall omit acute sinus inflammations and affections which tend to get well by simple forms of treatment. If vaccines are needed in such cases (more as a stimulant to the whole body than as a specific), we are content to rely upon any of the commercial catarrhal vaccines which are on the market.

From a theoretical point of view there are sufficient reasons for feeling that vaccines applied locally have a certain value over those given by hypodermic administration. In the usual vaccine that is prepared, a culture is taken from the infected part, sent to the laboratory and, after growing for a certain length of time on a prepared agar plate, the more important organisms are isolated, regrown, and the vaccine made from them. No account is taken of the minor organisms, the spores or the digestive products of the bacteria. How much value these products have in overcoming disease, it is hard to say but the recent studies of d'Herelle on Bacteriophage, have shown us that there are possibly ultra-micro-organisms which cannot be seen even by a super oil immersion lens, but which are capable of killing other bacteria. In fact, the whole idea of bacteriophage is that some such method takes place in the final dissolution of epidemics. I believe that this monumental work of d'Herelle is of sufficient importance to warrant further investigation.

That there are foreign products of bacteria which have an effect on tissues and continue to prevent a restoration of these tissues to the normal, has often been proved. The possibility that the bacterial origin of cancer may not have been discovered, may be due to the fact that we have not had means at hand for discovering the bacteria. Or is it not possible that there are extraneous products in cancer which may have a great deal to do with the spread of the disease? I mention this fact here because of the following case:

A man, age 50-odd years, a patient of Dr. Douglas Quick's, had been operated upon by him at the General Memorial Hospital for a carcinoma of the antrum, intraorally. Every vestige of disease had been removed and a few weeks later, he returned to his home in Porto Rico. Four months later he came back to the hospital complaining of terrific pain on the left side of the face. There was

considerable swelling over the malar bone, there was quite some exophthalmos and a small fistula had developed below the eye, which led down to the cavity from which the growth had been removed. The patient also had a great deal of discharge of mucopus from the nose, which suggested an infection of the ethmoid cells and sphenoid sinus on that side. Dr. Quick called me in consultation to ask whether I thought the sinus condition had anything to do with the symptoms from which he was suffering. An examination made me feel that there was no relationship between the two. After advising the cleansing of the nose with permanganate of potash douches, I suggested the use of an autogenous vaccine locally into the operated cavity and, with Dr. Quick's acquiescence, I took a culture from that part and had a vaccine made. At the end of three days, I examined the patient again. He was in a great deal of pain, the bulging of the eye had become more manifest and I felt that very little could be done medically and, that in all probability, Dr. Quick would have to remove the eye. However, I instructed the nurse in the use of the vaccine on pieces of gauze. At the end of a week, Dr. Quick informed me that the patient had greatly improved. The inflammation had subsided, there was scarcely any pus from the sinus and the pain had been much relieved. No treatment had been given other than the irrigations of the nose and the application of the vaccine. In another few days, the patient was apparently cured. I saw him six months later. He had gained in weight, there was no evidence of any disease and he felt entirely well.

I bring this dramatic case to your attention, hoping that it will suggest to you great possibilities for the future. Was the cure due to the destruction of the bacteria which aggravated the cancerous condition? Is it possible that carcinoma itself is bacterial in origin? Or is it possible that there are certain extraneous products in the whole culture which is used, which would bring about a cure? I ask these questions because I am as much at a loss as you are as to how the cure was brought about. Of one thing we are sure—that disease is not caused entirely by bacteria themselves, but by the chemical products which accompany the growth of bacteria. Thus we have the sapremias and fevers associated with various local bacterial infections.

In the laboratory we can prove that the extraneous products of bacteria may or may not have an effect upon the human body. We have been particularly interested in the action of the cultures which we have made and a few comments about them will be of interest. For the purpose of preparing the vaccine, a culture from the in-

fected area is made on a sterile swab and immediately sent to the laboratory. This is at once directed into a flask of sugar bullion and is allowed to grow for forty-eight hours, when it is killed by heat. Dr. Winslow's exact technique may be found in my former papers. In attempting to get cultures from the sinuses, one should be sure to make a direct application to the purulent material with his swab. Dr. Winslow found that the growth attained its maximum at the end of forty-eight hours, and from then on there was a tendency for the number of bacteria to diminish and for certain bacteria to predominate. Therefore it was found best to kill the organisms at the end of that time. The broth, with the contained bacteria and their secretions or excretions is used as the vaccine to be applied locally.

That the broth itself has certain altered properties after the growth of bacteria in it for forty-eight hours, was well proved. This broth was passed through a Berkfeld filter so that all the bacteria were removed. It was then reinoculated. No growth of the second group of bacteria could be obtained. The question naturally arises, "What happened to this broth? Had it developed certain bacteriophagic properties? Had it become bacteriocidal, or is it not possible that certain chemical changes had taken place in it which mitigated against the growth of organisms?" The question of bacteriophage is too theoretical for us to enter into a discussoin of it here. It is my belief that bacteriocidal properties developed which, in themselves were sufficient to increase the remedial properties of the vaccine. Of course it is very difficult to prove this fact. It is certain, however, that chemical changes did take place in the broth, among which were a change from an acid to an alkaline reaction and a loss of some of its nutrient properties.

The chief value of the vaccine, to my mind, is that instead of isolating and regrowing the predominating bacteria which may or may not be at fault, use is made of all of the bacteria obtained from the diseased part with any or all of the eliminative properties of these organisms. I think this in itself is a great step forward and makes me feel that there is much more possibility of cure in any given case provided the vaccine can be directly applied.

In a tabulation of the first two hundred cases in which the vaccine was used, there were eighty-eight cases of sinus disease, most of which were subacute or chronic. There were nine cases of antral infection, twenty-seven cases of ethmoiditis, and fifty-two cases of pansinusitis. Since this report, we have administered the vaccine in at least two hundred more cases, the proportion of sinus cases being about the same.

Although the determination of the organisms causing the infection is not of a great deal of importance because all the organisms in the culture are used, yet for scientific purposes we felt that it would be of interest to isolate the predominating bacteria. As might have been expected, the streptococcus group was most often seen either alone or in combination with other organisms such as the staphylococcus aureus, the micrococcus catarrhalis and the pneumococcus. It was not possible to determine in all cases the exact type of streptococcus, as cultural characteristics would not show on the growth in the sugar bouillon medium.

In old antral conditions and in cases of atrophic rhinitis, there was often a growth of putrefying bacteria which gave forth such an awful odor that it was impossible to use the vaccine in the form described. In such cases, we passed the broth culture through a Berkfeld filter, dried the bacteria and suspended them in a salt solution. In such cases, of course, we had eliminated a great many of the factors which we deem of importance, such as the chemical changes in the broth itself and we are not in a position at present to say whether this suspension was of as much value. It is our belief that it is not.

In analyzing the cases of sinus disease, one must bear in mind the fact that no judgment on the value of any remedy can be formulated in acute cases in which there is good drainage. The majority of such cases will get well when simple treatment is employed. We must therefore confine our observations to subacute or chronic cases. By the subacute cases we mean those in which there is a continual irritation of the nasal mucosa, a continual postnasal drip of mucopus, a general feeling of malaise, and pain either in the frontal region, over the antra or occipital. These patients have a tendency to clear up for a few days only to have a recurrence of the symptoms in a short time. By the chronic cases, we refer to patients with symptoms similar to the above in which there is no recession of symptoms. In this class belong the cases which have previously been operated upon with no good result and other cases in which we feel that medicinal measures should be used before advising operation. I am firmly of the belief that too many operations upon the sinuses have been performed in past years with no beneficial results and that the public are at the critical stage where they balk against the suggestion of operation unless they are actually suffering from acute pain.

You will note that in my classification in my former papers almost all the cases were spoken of as ethmoiditis or pansinusitis. The exceptions were nine cases of definite antrum infection. We did not

have one case of frontal sinus infection in which there was not some involvement of the other sinuses. This brings me to a most important point—that although one may have a definite infection of one or both antra unassociated with an affection of the other sinuses, it is seldom that one finds a chronic infection in the other sinuses isolated in one set of sinuses or on one side of the nose. If such a distinction is made, one may wonder why we classified certain cases merely as ethmoiditis. This was done as a matter of convenience and in this class were included, in the main, cases in which there was no active suppuration. In other words, these cases were of the hyperplastic type.

In the treatment of these cases, which include both the nonsuppurative and the suppurative cases, cleanliness is of the utmost importance before the vaccine is applied. We shall infer that the ordinary routine procedures had been used in the majority of these cases without any beneficial results. With the exception of the antrum cases, the following routine procedure was adopted:

Examination was first made of the nasal cavities after shrinking the mucosa with a mild cocain and adrenalin solution. The antrum and frontal sinuses were transilluminated with the Cameron lamp. The posterior portions of the nares were inspected with the nasopharyngoscope, particularly to see whether the sphenoid cavity or the posterior ethmoid cells were affected. In the majority of cases, X-ray pictures of the sinuses were taken, especially to determine the condition of the sphenoid sinus.

Our next procedure in all cases is the washing out of the nose with a suction irrigation apparatus. We find such an apparatus invaluable not only for diagnosis but for after treatment. By very simple means, we can rig up a suction bottle to a water pump and be able to measure the amount of suction by an ordinary indicator, which should never register over 5 pounds pressure. With a tube in either nostril, we are able to cleanse out the nasal cavities and the sinuses. After the first bottle has been filled, a second bottle is attached, and sometimes a third bottle, and then the washings in the three bottles are compared. In this way we not only determine the contents of the sinuses but also determine the efficacy of this form of treatment.

It may have happened that with this washing, we have so cleansed the mucosa that it is impossible to obtain a satisfactory culture. If pus is apparent before the washings, a culture should immediately be taken of it, on a sterile swab. Sometimes pus may be obtained by shrinking the mucosa and having the patient blow his nose on a piece

of sterile gauze or else dry suction may be used for a few minutes. If an unsuccessful culture is obtained on the first day, another culture may be made in the next twenty-four hours. The culture thus obtained should be immersed in the sugar bouillon at once. A delay of a few hours, during which the swab dries, may mean absolute failure in obtaining a satisfactory vaccine. The laboratory process for making the vaccine has been described in previous papers.

The method of applying the vaccine is very simple. The patient should receive eight office treatments in all at intervals of three to four days. In the interim, he should use the vaccine at home twice a day in the form of a spray. The mucosa of the nares should be mildly shrunk with cocain and adrenalin. The broth vaccine is placed on long strips of cotton or ½-inch gauze and packed into the nares, where it is allowed to remain from twenty minutes to half an hour. At the end of that time, the packings are removed and the mucosa sprayed with a mild antiseptic oil. If there is much actual pus in the sinuses or in the nose, it may be advisable to wash out the nose with the suction douche apparatus first. In other words, a local vaccine of this kind works best if it is applied to a cleansed mucous surface which is not too closely shrunk so that all the circulation is lost, which often happens if too much adrenalin or cocain is used. At the end of the eighth treatment, which means at the end of the fourth week, the patient ought to notice a decided improvement or cure.

Of course we are particularly interested in results and it is pleasing to me to state that these have been most gratifying. In the first sixty-nine cases reported, twenty-three were cured (33 per cent), thirty-eight were improved (55 per cent), and eight (12 per cent) were unimproved. One should remember that almost all of these cases had received other treatment at my hands or were in the hands of some other rhinologist previously and had not seemed to be benefited. As far as I know, not one of these cases had come to operation. Surely such results compare very favorably with the application of vaccines in other ways.

A special comment should be made upon the antrum cases. Here we have a cavity which not only can be made to drain through the natural opening but it is always possible to make a small opening through which a medicine can be directly applied and where such a medicine will remain for an indefinite length of time. In these cases, the antrum was first washed out and then a sufficient quantity of the vaccine placed into the cavity. This procedure may only have to be resorted to once or twice and then the rest of the treatment can follow the procedure outlined above.

In conclusion the following points should be stressed upon:

1. Cultures for the treatment of sinus conditions can be easily made and broth vaccines prepared from them in any well equipped laboratory.

2. The application of the vaccine is simple and has not the disadvantage of hypodermic injections, which is a particularly difficult procedure with children.

3. The vaccine should never be applied to the mucosa until it is first thoroughly cleansed.

4. In the majority of cases, the vaccine can be applied to the mucosa of the nose near the sinuses affected with the exception of the antra, into which the vaccine can be injected directly.

5. There is a possibility that the vaccine may have a specific effect in certain cases of malignant growth.

6. This vaccine differs materially from the usual vaccine, in that it not only contains all the bacteria from the diseased part, but the by-products of these bacteria and the chemically changed broth in which the growth took place.

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22 West 74th Street.

PRIMARY EPITHELIOID CANCER OF THE UVULA. PRESENTATION OF PATIENT; NO RECURRENCE EIGHT MONTHS AFTER OPERATION.*

DR. WILLIAM WESLEY CARTER, New York.

Mr. F., married, age 52 years, was referred to me by Dr. G. W. Carr, of Wilkes-Barre, Pa.

Family history has no bearing on present condition.

He has been a heavy smoker of cigars and a moderate drinker for many years. Has had a cough (bronchitis) for several years.

About the middle of May, 1925, two weeks before he consulted Dr. Carr, he had a sensation of roughness in his throat and some discomfort in swallowing and talking. He had some pain in the roof of his mouth.

Patient was seen by me for the first time on June 8, 1925. I noted that he was well nourished, but was in a state of great excitement. His blood pressure was 220.

Examination of the throat showed a general congestion. The uvula was enlarged and there was over the anterior surface, with the exception of the extreme tip, a grayish, ulcerated area, irregularly raised above the surrounding mucous membrane, and presenting a cauliflower appearance. This ulceration extended further up on the right side to the velum. The posterior surface of the uvula was not involved. There were no palpable glands, nor was there any evidence that the disease had spread beyond the uvula. The growth was hard to the touch and did not bleed when palpated. A tentative diagnosis of carcinoma of the uvula was made. The Wassermann test was negative. Dr. Lee M. Hurd was asked to see the case in consultation and he agreed with me as to its malignancy.

Patient was placed on a nonstimulating diet and in a few days his blood pressure was reduced to 160. He was then sent to the hospital and the entire uvula removed beyond the edge of the growth. Another piece of tissue was then removed, including the entire base of the uvula and a part of the velum. These specimens were sent to Dr. Ewing at the Cornell Laboratory. He reported that the specimen was an epithelioid carcinoma of a very malignant type, with a tendency to metastasize early. The second piece of tissue

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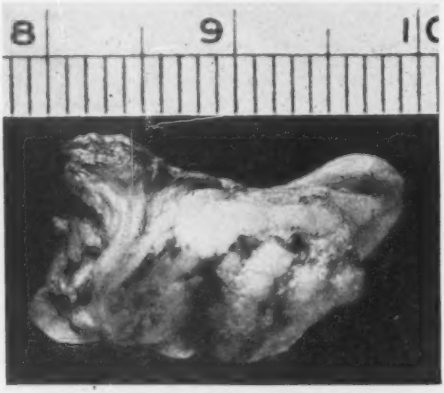
removed was apparently normal, showing that I had cut beyond the involved area.

Dr. Ewing's report in the case of Mr. F.:

Gross examination reveals a piece of tissue about 2 c.m. long by 1 c.m. wide, about the size and shape of the uvula. The tip is edematous and about ½ c.m. from this tip there is an irregular, warty mass about ½x¾ c.m., well outlined and reaching to the base of the uvula.

On *cross-section* there is a creamy white band arising from the surface, infiltrating into the submucus tissue. The second small piece of tissue removed shows no pathological change.



Microscopic examination: Section taken through the warty mass on the uvula reveals a fully malignant epidermoid carcinoma with typical pearls, many mitoses, extensive infiltration into the underlying tissue and some myxomatous degeneration in the connective tissue.

Note: The tumor is highly malignant, will infiltrate widely and will produce metastases early.

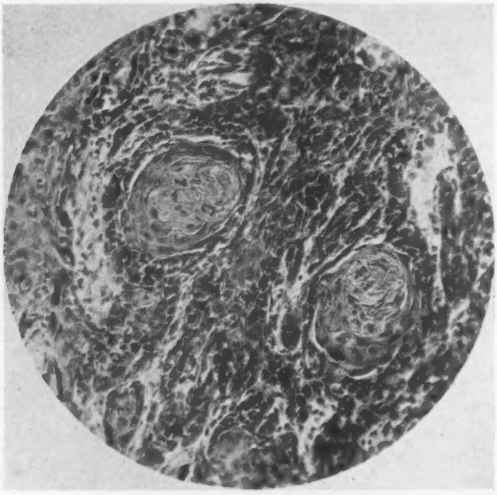
On receiving this report the patient was removed to the operating room and 18 radium seeds of ¼ m. Curie were introduced in a semicircle immediately above the edge of the soft palate; several seeds being concentrated just above the former site of the uvula.

The patient remained under observation for six weeks after the introduction of the radium. The ultimate effect of the radium was to leave a thin, hair-like line of cicatricial tissue across the soft

palate which did not interfere with its function either in speaking or swallowing.

At the present time, as you will note, the patient is in excellent physical condition and there are no signs of recurrence. The case came under observation very early and I believe that the disease has been thoroughly eradicated.

The radium seeds, which are almost as fine as a hair, and only about 1/16 of an inch in length, may be left permanently in the tissues with impunity. Their action is of maximum intensity during the first six weeks, but they are said to continue their activity for six months after introduction.



Among the interesting features of this case are: The extremely malignant type of the growth. The early stage at which it was discovered. The character of the treatment, while it did not destroy the function of the palate, has apparently eradicated the disease. However, the probation period is by no means over. I report the case at this time because the patient is here from a distant city and I am enabled to give you a clinical demonstration of the result.

Note: On May 20, 1926, nearly one year after operation, patient was thoroughly examined; he is in excellent physical condition and there is no sign of recurrence of cancerous growth. Palate functions perfectly.

THE REACTION OF THE LUNG TO THE ASPIRATED PEANUT.*

DR. J. D. KERNAN, New York.

It is the usual opinion that such foreign bodies, as peanuts, when aspirated into the lung invariably produce an extremely severe reaction and are usually fatal. That has been my impression, gathered from the reports of cases. It has happened, however, that I have had eight cases of aspirated peanuts. Of these cases, in five, the foreign body was removed; in three, it was not removed, and yet these three cases survived many years after the aspiration of the peanut. The eight cases form a rather interesting series, ranging from two days after the aspiration of the nut to twenty-seven years.

The first case is shown here. The patient, a child, age 14 months, had been given a peanut to eat and aspirated it, two days before admission to the hospital. Her symptoms consisted of a spasmodic cough, a rise of temperature to 100,° and rapid pulse and respiration. There was a slight dullness over the right middle lobe, with râles over the right middle and upper lobe. The slide shows very little change in the right lung. Most of the shadows in the picture are on the left side. I am told by the Roentgenologist that that is due to the poorness of the plate; there is a slight flattening of the diaphragm on the right side.

At the end of two days there had been no inflammatory reaction to the peanut visible in the X-ray plate. When it was taken out, the temperature fell to normal, respirations 24, pulse 120—two days after the procedure.

The next case was a baby, age 2 years, who aspirated a peanut and was sent to the Lenox Hill Hospital on Jan. 1, 1925, four weeks after the accident. The symptoms were choking and coughing, which had persisted for a month. The temperature on admission was 102.8,° breathing irregular; the left side was dull; with hyperresonance on the right side and distant breathing on the left. The X-ray showed an almost completely drowned lung on the left side. The peanut was removed, and in twenty-four hours the temperature

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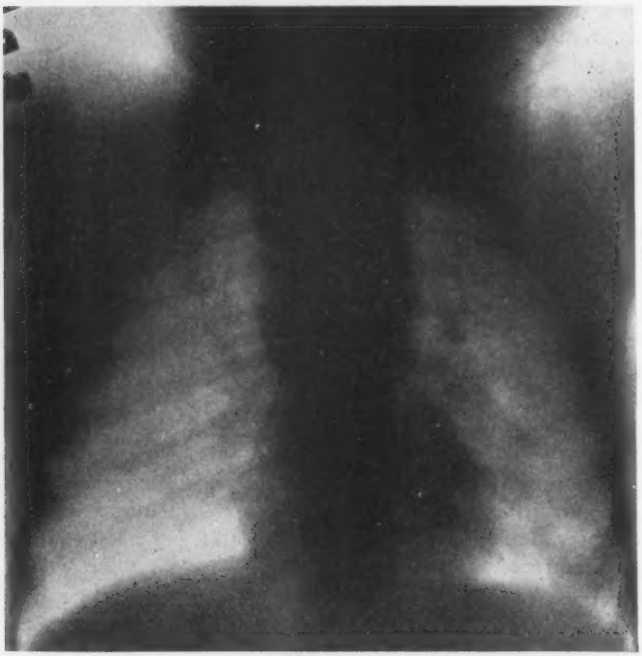
was 99° and the baby practically well. This child had not appeared as sick as one might have expected; although it had a cough and fever, it was getting up on the bed and playing, and was very slightly depressed for the amount of trouble in the chest.

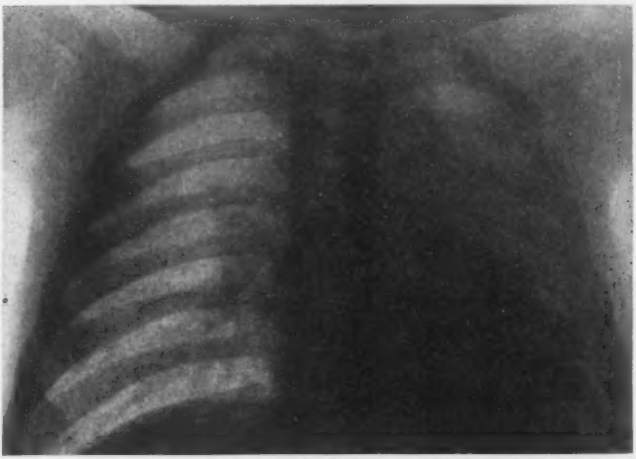
This picture was taken at the end of a week after the removal of the peanut. You will see that the lung condition was rapidly clearing up, and the child was discharged practically well at the end of a little over a month after the peanut was aspirated.



Case 1.

The next case was that of a baby age 18 months. This picture was taken two months after the aspiration of a peanut. When eating he had choked and coughed, and then began to cry; his symptoms were a spasmodic cough, fever for half a day, and after which his temperature came to normal; except for one short rise, it remained normal. Two days before admission to the hospital, he spit up a spot of blood, but he was up and around the house playing,

and showed very few symptoms. The signs were diminished breathing at the angle of the scapula, slight dullness, most marked at the angle; the breath sounds were diminished over the same area. The X-ray picture shows the condition of the lungs two months after the aspiration. There was no such reaction as in the case where the peanut was a month in the lung. I had a curious time removing that peanut. As I pushed the tube down it ran into a ring of thickened mucous membrane forming a stricture, and as the tube pushed through, the baby coughed and something hit me in the eye, which proved to be a small piece of the peanut. I took another look, and there was another piece bobbing up and down—that was another bit of peanut, etc. This was repeated until six small pieces of peanut had been either coughed up or removed by the forceps. Probably



Case 2.

the mass had been bound together by mucus and so aspirated. The swelling of the mucous membrane had kept it altogether. At the end of a week, the X-ray showed no consolidation in the lung, and there were no signs over the lung.

The next case was an exceedingly interesting one—a child, age 11 years, who seven years before had aspirated a peanut. The attempt made to remove the peanut at that time had failed. She had a cough with very foul sputum ever since. Examination showed a well nourished and developed child, with cyanotic cheeks

and clubbed fingers, etc. The left side of her chest was poorly developed. A large amount of foul sputum poured out of her mouth in streams. The picture shows the complete destruction of the left lung. It had turned into a honeycomb of fibrous tissue with empty spaces scattered through. Here is a gathering of fluid in one of the larger spaces at the base. I bronchoscoped her a number of times, and gave her up in despair. There was no improving her at all. She finally went back to Norway.

The next case was a man, age 27 years, who had aspirated a peanut twenty-three years before admission, when he was age 4 years. He had had pneumonia and empyema, which was opened, a pleurisy, etc.

In March, 1925, he had an hemoptysis and was admitted to the Lenox Hill Hospital. On admission his whole left chest was solid to percussion and the breathing was distant bronchial breathing. A picture of the lung, as in the previous case, showed a solid lung with cavities all through it, and the mediastinum pulled over to the diseased side. We started to bronchoscope him without much hope of success; curiously enough he began to improve, and from spitting up 400 c.c. of foul sputum a day he now has but 80 c.c., without odor, has gained 15 to 20 pounds in weight, and his clinical symptoms have disappeared.

I present these cases to show: first, a series of cases of aspirated peanuts ranging from one month's duration to twenty-three years; and, in the second place, to bring out the point that the patients don't all die; third, to show that even though they don't die, the lung may be completely destroyed in practically every case.

120 East 75th Street.

THE PREVENTABLE DISEASES OF CHILDHOOD.*

DR. WM. B. CHAMBERLIN, Cleveland.

Medicine, whether it concerns human beings or animals, must recognize two aims: first, the cure of disease, and second, its prevention. Since human beings, as well as animals, are mortal, the first of these must ultimately end in failure. The second, medicine has in part answered and bids fair to answer even more successfully in the future. The longevity of the race, by modern treatment and prevention, has been increased by at least ten years. The diseases of the gastrointestinal tract, which since time immemorial have been the scourge of armies in the field, have been practically eliminated, as shown by the records of the Russo-Japanese war, of our troops when congregated on the Mexican frontier, and by the records of the great war. Diseases of the air passages, however, still claim their toll. Proper water control has practically eliminated typhoid fever from our larger cites, so that the responsibility for its continued existence in sporadic instances now lies with the lowly and modest oyster.

In speaking of the preventable deafness of childhood I wish to state, and shall attempt to prove, that, barring congenital deafness and the occasional fulminating forms of the exanthemata, practically all deafness of childhood *is* preventable and, as a sequence of this, most of the deafness of adult life. For convenience in the discussion I shall divide my subject into three parts: first, causes; second, results; and third, prevention, which will of necessity embrace treatment.

1. *Causes.* At the beginning of his chapter on the causes of acute tympanic disease, Kerrison speaks as follows: "The conditions which predispose to, or excite, middle ear inflammation may give rise to the mildest type of tympanic congestion or to the severest form of suppurative otitis media. That is to say, it is not possible, in the light of our present experience and knowledge, to speak dogmatically of one group of etiological factors as producing the milder ear lesions and of another as responsible for the severer types of tympanic disease." Here again we have to do with the virulence of the organism and the susceptibility of the individual.

Among *predisposing* causes, the existence of adenoid tissue in smaller or larger amounts in children plays a most important role. The nasal obstruction and middle ear catarrh which an adenoid mass

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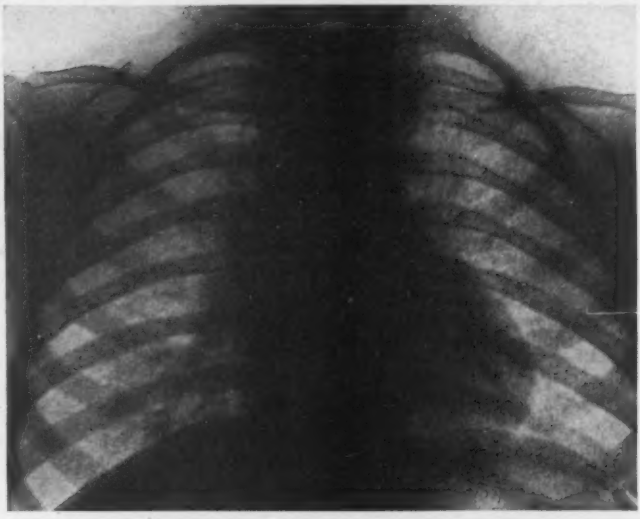
may cause is not dependent upon its absolute size, but upon the relationship which the mass bears to the nasopharyngeal space which it occupies. So, a small adenoid mass may cause great damage when housed in a small nasopharynx—a large adenoid mass little or no damage if the nasopharynx is roomy. Gulliver was a pigmy among the giants, but a giant among the pigmies. That children of the poorer classes, where there is accompanying ill nourishment, are much more disposed to adenoids and consequent middle ear involvement, is the experience of everyone who is familiar with dispensary practice in our larger cities. An adenoid mass which is causing nasal obstruction, frequent colds and middle ear suppuration, should be immediately removed, no matter how young the patient.

Children are much more susceptible than are adults to middle ear infection, not only on account of the presence of adenoid tissue, but also on account of the anatomical differences in the Eustachian tubes, the tube in the child being shorter, more patulous and lying more horizontally. In addition to these we have the exanthemata. Nasal obstructions such as spurs, deflections, hypertrophies and polypi, predisposing factors in adult life, play little part in childhood.

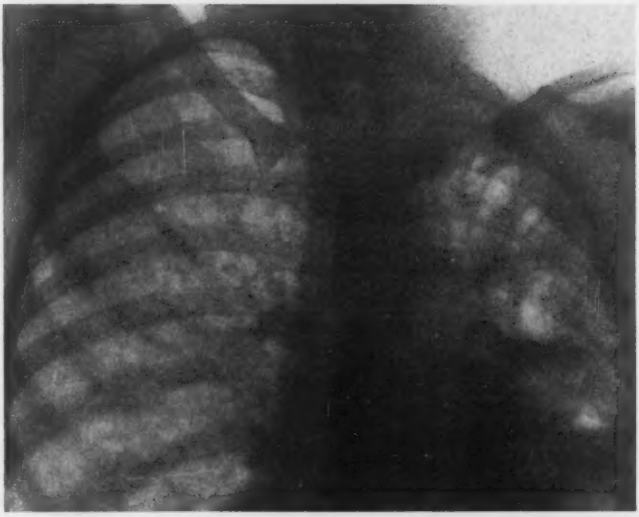
As *exciting* causes probably the first place should be given, not only in the child, but in the adult as well, to the common cold, or acute coryza. The mucous membrane which lines the Eustachian tube, as well as that which lines the middle ear, is merely an extension of the mucosa lining the nose and nasopharynx. When we realize the enormous congestion which characterizes an acute nasal and nasopharyngeal infection, as well as the profuse secretion which both accompanies and follows it, our wonder is, not that there should be an occasional middle ear involvement, but that middle ear involvement should not complicate every acute coryza. The presence of even a relatively small adenoid mass in children renders middle ear involvement in an acute coryza all the more probable.

At least 10 per cent of all cases of measles and scarlet fever, and 5 per cent of all cases of diphtheria have some middle ear involvement. These percentages are from the statistics of the Willard Parker Hospital. Routine examination of all cases would undoubtedly render this percentage much higher. Grip, or influenza, is also a common cause of middle ear suppuration.

Mumps, although it rarely leads to a middle ear suppuration, occasionally causes a complete deafness, of the internal ear type. The wife of one of my professional colleagues has a complete unilateral deafness, the result of mumps. A boy age 14 years, who came under my observation with a bilateral middle ear suppuration, had



Case 3.



Case 4.

an almost complete deafness in the left ear with still fair hearing in the right. An acute parotitis caused a complete loss of hearing in his better ear.

Among *external* causes exposure to cold, with accompanying wet feet and improper clothing, plays an important part, in children as well as in adults. The practice of some physicians, as well as some pediatrists, in advising the nasal douche should be mentioned only to be condemned. Whereas the nasal douche is helpful in removing nasal secretion in children too young to remove the secretion by blowing, it involves too great a danger of carrying infection directly into the middle ear cavity. Improper blowing the nose in older children may accomplish the same result. The nose should always be blown by making compression on one side only, thus leaving the opposite side entirely free.

Our modern life has not only become more complex, but man, as well as the child, has certainly become more amphibious. MacMillan states that none of the Esquimaux can swim. In many visits among the Indians of the Canadian woods and those of the southwest I have rarely seen an Indian use water for other purposes than drinking or navigation. Yet most of our modern children are daily in the ponds and streams which nature affords in summer and the children of the more well to do in artificial pools during the winter season in addition. Swimming impairs the hearing; first, by the sudden and forcible impact of the water against the tympanic membrane in diving; and secondly, by its entrance into the middle ear cavity when it has gained access to the nasopharynx. The former can be obviated by plugging the ears with cotton or some variety of rubber stopper; while the latter danger can partially be removed by proper efforts to prevent the water from entering the nose and nasopharynx. At the Lakeside Clinic we always expect the seasonal crop of external otitis and middle ear infections when the young man's fancy lightly turns to thoughts of love and the young boy's to the mouth of the sewer which opens on the lake shore just adjacent to the hospital.

2. Results. There are four symptoms of which a child, or an adult, with ear trouble will complain: pain, deafness, discharge and tinnitus. Unfortunately the child, as a rule, complains only of the first. An infant crying and fretful, especially with fever, always deserves an examination of his ears. That the small baby always, or usually, puts his hand to his ear, if pain originates from that source, is an exploded fallacy. Nature rarely makes diagnosis for the physician so simple. Far be it from me to insist that the family physician should be an expert in examining the middle ear or

that if he isn't he should call an otologist. Frequently there isn't such an animal within 50 or 100 miles. I have lived in some of the out of the way places in our country and after hearing both sides of the story my sympathy goes out to the family physician,—and sometimes to his patients also. But I do insist that an ability to examine the ears should be a part of the armamentarium of every pediatrist. Fortunately, in Cleveland at least, I feel quite sure that such is at present the case.

Deafness with a child is an elusive symptom. Frequently its onset is mild and its progress slow. When the onset is sudden even an older child may make little or no complaint. Often a child is accused of inattention when spoken to when, as a matter of fact, the child does not hear. The differential diagnosis between deafness and inattention is often a difficult one for a mother to make. The child has a single track mind—that is to say, he is usually completely absorbed in the one thing which he is doing and it is difficult to divert him. Possibly many of us realize the misfortune in that this characteristic should not have persisted into adult life. In small children comparative hearing tests cannot be made. Like the shadow tests for the eyes, impairment of the hearing is usually shown by fairly definite changes in the appearance of the tympanic membrane.

Discharge, unless pain accompanies it, rarely annoys a child. Unfortunately it rarely annoys a parent of the poorer classes, and sometimes not even the parents among the more well to do. A discharging ear is always a dangerous ear. Any physician who regards it in any other light is either grossly ignorant or derelict if he fails to so inform the parents.

Tinnitus, unless extremely severe, is never complained of.

3. Prevention and Treatment. What then are the physical findings which may lead to the diagnosis of temporary deafness in children and to its ultimate cure. Among the first may be mentioned a drawing in of the tympanic membrane, with accompanying prominence of the short process. This is due to a closure of the Eustachian tube and the consequent absorption of the air in the middle ear cavity. A partial vacuum is the result. The tympanic membrane is not sucked in, but is pushed in by the pressure of the surrounding air. There is a lack of balance in the air pressure on the inner and outer sides of the tympanic membrane. Even in young children this balance may be restored by inflating the ear by the Politzer method. In older children the Eustachian catheter may also be used. In addition to the so-called retraction, there may be present a dark hair-like line, stretching across the tympanic membrane. This is nothing more or less than the upper level of an effusion within

the middle ear cavity, as shown by its change in position on moving the head. Here also inflation by the Politzer method will be effective. If the Eustachian tube is not opened, permanent retraction of the membrane and so-called catarrhal deafness is likely to result. If the fluid is not removed an adhesive middle ear process may take place.

Again we may notice some congestion of the membrane, together with a marked injection along the hammer vessels. Such a condition may mark the beginning of a suppurative otitis media. Its presence is usually accompanied by pain. Frequently a solution of 30 drops of carbolic acid to the ounce of glycerin, dropped into the ear as hot as can be borne comfortably, will relieve the pain and possibly abort the otitis. The ear should be observed afterwards to see that no retraction of the membrane takes place.

In the severer forms of infection the normal concave surface of the tympanic membrane is replaced by a surface which is *convex*. In addition there is usually more or less redness, together with intense or even agonizing pain, as well as fever. Such a condition calls for prompt paracentesis. The discharge at first is always serous, changing later to mucopurulent and mucoid. The family, as well as the attending physician, occasionally ask whether pus follows the incision. Pus on incision always indicates that the paracentesis has beed made *too late*. In other words, if the incision is made as early as it should be made we should always get serum and never pus. Not infrequently, however, it is impossible to see the patient before pus has formed. The rapidity with which marked bulging or even rupture may follow the initial pain is at times astounding. In scarlet fever occasionally we may meet with a fulminating form of infection, where complete necrosis and loss of the tympanic membrane follows quickly upon the initial symptoms, even before a paracentesis can be made. Such a condition leads to a permanent perforation, usually of large dimensions, with irreparable impairment of the hearing.

The discharge from an acute middle ear suppuration usually ceases in from one to three weeks. Following the cessation of the discharge the ear should be inflated regularly in order that retraction and adhesions of the membrane do not take place. If the discharge persists for a longer period, even in the absence of all other signs, involvement of the mastoid process should be suspected. A discharge from the middle ear which persists unabated for four, or at most six, weeks is almost positive indication of mastoid involvement and demands that the mastoid cells be exenterated. Such a persistent discharge, unless relieved by mastoid operation, carries with it

grave danger of intracranial complications and is almost certain to lead to permanent impairment of the hearing. The opening of the mastoid process in such cases has been followed for many years in our clinic and private practice at Lakeside Hospital. We have as yet operated upon no patient where we felt, subsequent to the operation, that such operation could have been avoided. The simple mastoid operation, in uncomplicated cases, carries with it little or no risk to the patient. Failure to operate carries with it grave risk to life and almost certain deafness of a greater or lesser degree.

I cannot bring this paper to a close without a word in regard to chronic suppuration of the middle ear. Here the hearing is already irreparably impaired. If the suppuration is bilateral, as is not infrequently the case, the condition, as far as hearing is concerned, is indeed tragic. In addition, whether the ear be dry or not, there is an ever present danger of serious intracranial complications. A chronic middle ear suppuration is an absolute bar to the obtaining of life insurance in most of the better companies, and to military service as well. Some, during the late war, did not consider the latter a disadvantage.

In the vast majority of instances a chronic middle ear suppuration is an absolute evidence of ignorance and neglect; on the part of the *physician*, if he has failed to inform the parents; on the part of the *parents* if they have failed to follow his advice. Continued discharge from the middle ear, in the absence of any other signs, is conclusive evidence of mastoid involvement. If the mastoid process is thoroughly exenterated and drained at the proper time, resolution is almost certain, a chronic suppuration is averted, and the consequent danger to life and hearing removed. The only possible relief for a chronic suppuration is the radical, instead of the simple, mastoid operation. The radical mastoid operation is performed with only one end in view—the prevention of intracranial complications. With the hearing already impaired beyond the possibility of relief, our one aim here should be to stop the course of the infection before it has spread to the lateral sinus, the labyrinth, or the brain. It is an operation to *conserve* human life and should be performed before complications have arisen.

In so brief a paper I realize that many important points have received scant attention. If I shall have convinced some that much of the deafness of childhood, like most of the blindness, is preventable and that for this prevention the physician is, in the last analysis, largely responsible, I shall have achieved my purpose.

Osborn Building.

"AN HONEST CONFESSION IS GOOD FOR THE SOUL."

DR. DOUGLAS MACFARLAN, Philadelphia, Pa.

It would seem a platitude to say that inasmuch as we are otologists we are interested in, or should be interested in, two great endeavors, — the protection of life from the complications of ear infection, and the protection, preservation and improvement in hearing. Yet such a platitude expressed, will bring to mind the fact that in one of these fields, that of the problems of the deaf, little progress, or even little effort has been made by medical men. The great accomplishments by the deaf themselves — by this I mean the organizations of the deaf — tell us at least that we have fallen somewhat short of their achievements. They have had to do for themselves what we did not do. We see in such literature as the *Volta Review* how the deaf are organizing into groups for self help; we read their articles of encouragement and helpfulness, and we see a list of lip-reading classes scattered throughout all the larger centers. To what extent are aurists taking part in the work? We discover but a few medical men among the organizations. Fortunately this small representation is strong with such men as Shambaugh, Richardson, Goldstein, Phillips, Berry, Kenyon and Harold Hays.

If we seek to see who is doing the scientific work with the deaf problem, we find for the most part physicists, physiologists, psychologists and engineers — Seashore, Pohlman, Dayton C. Miller, Bunch, Kranz, Harvey Fletcher and Sabine. The few medical men include Dean, Crockett, Shambaugh, Mackenzie, Sonnenschien, Fowler, Guttman and Jones.

This means that the majority of aurists are not "in line", and many are really not interested in that which the hearing public deems most important.

We may well ask ourselves why there is not more enthusiasm for such subjects as the testing of hearing, the fixing of standards of examination, the establishment of some practical grading of degree of deafness, or the improvement of instruments for the aid of hearing. We may well quiz ourselves as to why we do not check up improvement or retrogression in deaf cases, using accurate and rational hearing tests. How many of us are using tests that are intelligible to ourselves or to others? How careful are we in making tests?

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How often does the subject of the deaf patient's psychology come to our minds, and how often do we speak to our deaf cases upon this psychology, attempting to help them adjust themselves? I fear from our answers to these questions that our interest appears to be decidedly casual and even careless. I fear that we who have hearing do not realize what it is to be deaf; for certainly if we did our efforts to help would be greater.

We are open to some criticism upon this matter, as it is naturally expected of us that we should know more about this feature of our "trade".

Perhaps we have a few good reasons to explain these delinquencies. It is the fault of the patient that in his stage of incipient deafness we cannot help him under careful and continuous treatment. Deafness is like a habit that grows slowly; it has insinuated itself before its presence is appreciated. Again, like a habit, it is as hard to cure. To those with slowly progressing deafness the forceful comparison of hearing to deafness is only weighty when contrast is sharp, or when, all too late, the patients can themselves review the extremes of their condition. The psychology of the patient is one of slow adaptation. For example, here is a man who comes in with almost total deafness, which had its beginning ten or fifteen years ago. He has gently and unconsciously accepted his affliction. It is almost safe to say that it is useless to offer him the best service you can give—free—if he will come to you conscientiously for one year, for six months, or even for two months. The same may be said of the cases of incipient deafness. They cannot be made to realize.

Yes, the deaf man's day is long; his disappointments are many. He develops his mental attitude very naturally, and unless he feels the stimulus so happily and heroically thrown out by that small band of deaf who have not "given up"—he drops into a stolid complaisance like a squirrel in a cage. Soon he knows no other life; the edge of disappointment is dulled—and he accepts all. It is perhaps too late for him to work back the path down which he came. It is perhaps too late for an aurist to improve his hearing.

This is a hopeless prospect then, for which we are not to blame. We cannot help these cases physically, yet we can help them enormously by a word of courage, by unuttered sympathy, and by that most valuable suggestion to get in touch with the organized deaf. In the lip-reading schools and in the various city "leagues" they will find companionship and understanding, and they may, by their associations and by their efforts, learn to lip-read with wonderful accuracy.

Best of all, they will lose their introspection and their sensitiveness. The world will open up again to them. Aurists have this one excuse —the lack of co-operation of the patient. It is hard to find any further reason why we do not get busy with the deaf problem.

I see no reason why aurists should not at least sit in judgment upon the countless instruments that are sold as aids to hearing. Thousands of dollars are wasted yearly by undirected patients. Many of these instruments are as useless as they are expensive. The prescribing of "deaf sets" is done by laymen totally ignorant of what the patient has a right to expect. They are dispensed on the "trial and error" principle.

What do the oculists think of the vendors who sell glasses on the sidewalk?

It may not be possible for aurists to invent what will be the best aids to hearing, but at least they can help in their intelligent selection.

I see no reason why aurists should complain that they are too busy to make a careful examination of hearing. When one reaches a stage of average efficiency and intelligence in examining hearing, it takes no longer than a half hour to get an accurate idea of the hearing status. Merely for purposes of a record the time is well spent, and does not necessarily have to be repeated at each subsequent visit. The oculists take as much time over their examinations and do not consider it time wasted.

I see every reason why aurists should know of the work of the American Association to Promote the Teaching of Spech to the Deaf. They should know of the nearest Speech Reading League or School. They should keep on file a copy of the *Volta Review*. They should learn thoroughly the hearing tests, should establish a dependable technique, and then should use the tests.

In short, as aurists we have an interest in hearing; let us take an interest in hearing.

1805 Chestnut Street.

TUTOCAIN; A LOCAL ANESTHETIC IN RHINO-LARYNGOLOGY.*

DR. O. D. CUNNINGHAM, Iowa City.

The methods used by the ancients for the production of local anesthesia remained in vogue until the latter half of the nineteenth century. The discovery of the hypodermic needle by Wood in 1853 marks the beginning of modern local surgery. For twenty years or more cocain was the only drug used to any considerable extent and during this time cocain anesthesia and local anesthesia were synonymous terms. The large number of deaths occurring through the use of cocain by the skilled and unskilled gave impetus to the search for a drug less toxic in its effects. This led to the discovery by Giesel in 1891 of tropococain, a second alkaloid from the leaves of the cocoa plant. This was followed by the production of eucain, holocain, those of the orthoform group, stovain, novocain (procain) and others too numerous to mention. Of these latter drugs, novocain has the least toxic action and continued use over a period of years has shown that it well deserves the faith that is put in it.

It is not a sound policy to forsake the familiar, dependable local anesthetic for every innovation. However, the results reported by European investigators who have used tutocain appear to warrant its laboratory and clinical investigation here.

During the past two years this drug has found rather extensive use in the continental clinics for surface, infiltration and conduction anesthesia in the fields of rhino-laryngology, general surgery, urology, gynecology, ophthalmology and dentistry, and to a more limited extent in otology. Schuleman,[5] Wagner,[10] Hirsch[27] and E. Watson-Williams[11] were among the first to study the minimal effective concentrations and toxic dosages for animals. Because of the variable results and the limited scope of the experimental work, a laboratory study of the comparative effects of tutocain, cocain and novocain for corneal anesthesia and toxicity in animals, and at the same time a trial of its clinical use were attempted.

The investigations of the European writers have, very logically, followed four lines: *1.* pharmacology; *2.* toxicity when injected subcutaneously or intravenously into guinea pigs, rabbits, mice and rats; *3.* corneal anesthesia to the eyes of humans and of cats and rabbits—a test of surface anesthesia; and *4.* intracutaneous wheal anesthesia—a test of infiltrative power.

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*From the Department of Head Specialties, State University of Iowa.
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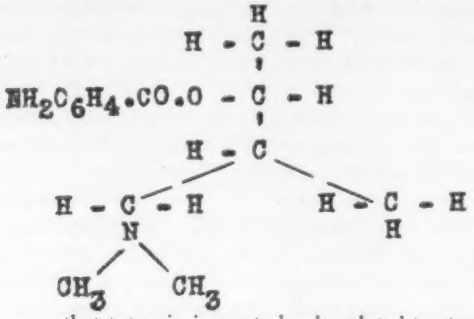
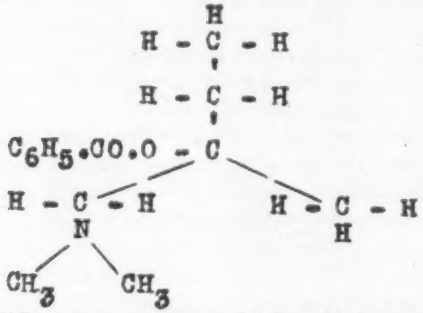
PHARMACOLOGY.

Tutocain is the hydrochlordi of p-amnoibenzoyl-a-di-methylamino-b-methyl-g-butanol, as shown in the formula below, in comparison with the bases of two other local anesthetics of the ethanol and iso-amyl, or butanol series. The structural formula of tutocain is as follows:

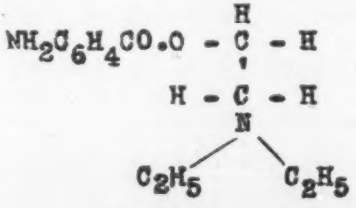


It will be seen that tutocain is most closely related to stovain, which is as follows:



Tutocain differs from stovain in having the benzoyl nucleus attached to the second C atom instead of the third, and in having an amino- group attached to the benzene rng in the para- position.

Novocain, which belongs to the ethnol series, has the following structural formula:



Tutocain contains two asymmetric C atoms. Its molecular weight is 286.6 and its alkaloid content 87.3 per cent. It has been isolated by Schuleman[5] in two racemic forms. It occurs in microscopic crystals that form a white powder, neutral, optically inactive, bitter to taste, and readily soluble in water to 15.4 per cent at 20° C., 23 per cent at 30° C., and 12.2 per cent at 10-11° C. It is clear and opalescent in solutions over 3 per cent. It soon becomes brownish if allowed to stand in solution with adrenalin. It is reported to withstand decomposition on being boiled or autoclaved for a short time, but to decompose in the presence of alkalies. The powdered form is quite stable; some samples used in this study were potent after eleven months. It is said to remain active in solution for several days if kept in a corked bottle with one part per 5000 of salicylic acid. For practical purposes it should be made up the day of its use and if adrenalin is added, this should also be done immediately before use.

The sale and distribution of tutocain is not regulated by the Harrison Act.

The cost of a tutocain anesthesia for a tonsillectomy is approximately 10 cents, and of the cocain-novocain combination for the same operation approximately 20 cents.

TOXICITY.

The toxicity of tutocain varies greatly, as does that of other local anesthetics, depending upon *1*. the place of injection; *2*. the rapidity of injection; *3*. the concentration of the solution used; *4*. whether adrenalin is instilled with the drug; and *5*. the species of the animal.

The method of subcutaneous injection, which appears the nearest approach to the clinical procedure, permits the largest lethal dose. Next in toxicity comes the mesenteric intra-venous method which allows, as does the subcutaneous method, a detoxication by the liver before the vital centers are overpowered. This detoxication allows many times the fatal doses of the substitutes of novocain (not of cocain) to be injected subcutaneously and also permits the usual lethal intravenous doses to be tolerated. This is not the case with cocain. The rapid intravenous method lessens markedly the lethal dose and hastens the onset of toxic symptoms.

The rapidity of injection, concentration of the solution, and the use of adrenalin locally are factors governing the rate of absorption and the possibility of detoxication without dangerous blood concentrations of the invading drug. For example, Eggleston and Thatcher[2] gave 408 mgm. per K of novocain to the cat or rabbit over a 3½-hour period without apparent toxic effects, but found 40 mgm. per K fatal on rapid injection.

Animals react differently to toxins and shock. Smitz and Lavenhart[6] were able to give rats 1600mgm. and mice 900mgm. per K of novocain, and cats but 450 mgm.

Twenty-four guinea pigs were used in this study to test the toxicity of tutocain as compared with that of cocain and novocain. The pigs, except the last one shown in Table II, were used in such a sequence that at no time was any particular drug administered to an animal a second time, thus avoiding the element of tolerance. Each pig was snugly wrapped for preparation and injection, the abdominal hair closely clipped, the skin washed with alcohol and the needles and syringes sterilized. Sterile distilled water was used in preparing the solutions, none of which were boiled. Except in three cases, as noted below, the solutions were 1 mgm. to 1 minum, or approximately 1½ per cent. The guinea pig injections were made into the loose tissue under the abdominal skin and were fairly rapid so that wheals were formed. If some of the solution went into the peritoneal cavity, or was lost, the experiment was discarded. Each animal was weighed and the exact dosage in mgm. per K calculated at the time of injection. Constant observation was made of each, except as indicated in the tables, where the symptoms were prolonged and the observation discontinued until the following morning. A protocol was made for each experiment, containing notations of the progress as occasion required.

A representative protocol is given in Table I which shows the typical train of symptoms and findings for a toxic, nonlethal dose of tutocain.

TABLE I.

TIME

4:03: Guinea pig No. E, weight 300 gm., given 51 mgm. tutocain subcutaneously (170 mgm. per K).

4:10: Refuses to walk.

4:14: Episthotonus. General spasticity. Sways from side to side, front feet braced.

4:16: Slowly falling to side. Rear legs spreading.

4:17: Prostrated. Respirations very rapid and shallow.

4:22: Lying in spastic paralysis. Occasional clonic convulsion.

4:25: Marked spasticity. Generalized convulsions. Artificial respiration.

4:30: Powerful convulsions. Dyspnea. Exophthalmus. Condition critical.

4:40: Flaccid at times. Small emesis. Artificial respiration discontinued.

4:45: Convulsions. Somewhat spastic. Shallow breathing.

4:55: Respirations regular, deeper and slower.

5:00: Raises head. Eyes brighter. Emesis.

5:15: Up on front legs; drags hind legs.

Next a. m.: Apparently normal.

Artificial respiration was instituted and compound spirits of ammonium put to the nose when death seemed imminent. With the cessation of respiration, camphorated oil was sometimes injected and in one case adrenalin, but without appreciable benefit. Artificial respiration, however, undoubtedly prolonged life in some cases.

The toxic course of symptoms for each of the three drugs was essentially the same except for minor variations of behavior. The cause of death appeared always to be spastic respiratory muscle paralysis, suddenly becoming a limpid paralysis in fatal dosages with deepening cyanosis. With the tutocain and cocain fatalities, the thorax was opened after the cessation of respiration. With the former, the heart continued to beat for an average of almost five minutes, with the latter almost eleven. It appears, therefore, that the treatment for poisoning with these drugs should be primarily artificial respiration and the administration of respiratory stimulants and oxygen. Better still, appear to be cerebral inhibitory drugs, such as barbital-paraldehyde, recently demonstrated in cocain poisoning by Tatum.[9]

Tables II, III and IV show the results of subcutaneous injection of toxic doses of the three drugs.

TABLE II.

Toxicity of Tutocain to Guinea Pigs, Subcutaneously.

Dose in mgm. x K	Onset of symptoms in min.	Duration of symptoms	Outstanding symptoms	Result
80	12	40 min.	Weakness rear legs; tremor; sluggishness; tachycardia.	Recovery
80	12	50 min.	Same.	Recovery
80	15	10 min.	Only more quiet than usual.	Recovery
90	13	43 min.	Wabbling gait; rear paralysis; remains on back when so placed.	Recovery
90	12	45 min.	Sprawling of hind legs; lateral tremor; muscular tonicity.	Recovery
120	15	1 hr. 43 min.	The above, plus spastic generalized paralysis and mild tonic convulsions.	Recovery
130	11	1 hr. 35 min.	Listlessness; polpnea; hypertonicity; spastic paralysis.	Recovery
140	15	1 hr. 33 min.	Weakness; clonic motions and spastic paralysis left hind leg.	Recovery
150	12	*	Exophthalmus; spastic paralysis; clonic convulsions; cyanosis.	Recovery

TABLE II (CONT.)

Dose in mgm. x K	Onset of symptoms in min.	symptoms of Duration	Outstanding symptoms	Result
160	15	*	Rear paralysis; generalized spastic and clonic convulsions; cyanosis.	Recovery
170	7	*	Same as above, but more marked; emesis; episthotonus.	Recovery
180	5	35 min.	Same, with more respiratory difficulty; incontinence.	Death
180	10	1 hr. 25 min.	Spastic bracing of front legs; episthotonus; generalized spasticity and convulsions.	Recovery
180	5	1 hr. 45 min.	Same, with rear paralysis.	Recovery
190	7	*	Episthotonus; fixed gaze with divergent squint; tremor; violent convulsions.	Recovery
200	8	40 min.	Spastic paralysis; powerful clonic-tonic convulsions; rigid chest; cyanosis.	Death
200	5	40 min.	Same.	Death

*Normal on following morning.

TABLE III.

Toxicity of Cocain to Guinea Pigs, Subcutaneously.

Dose in mgm. x K	Onset of symptoms in min.	Duration of symptoms	Outstanding symptoms	Result
20 (3%)	25	8 min.	Early rear paralysis; excitement; violent generalized convulsions, clonic, later tonic; respiratory muscle spasticity ending in a limpid paralysis.	Death
30	20	*	Wabbling gait; rear paralysis; episthotonus; clonic and tonic convulsions; shallow irregular respiration; rapid masticatory motions of jaw; cyanosis.	Recovery
30	5	1 hr. 10 min.	Early sluggishness; then excitement and spasticity with episthotonus; convulsive kicks; rear paralysis.	Recovery
40	2	6 min.	Loss of balance; excitation; rear paralysis; mydriasis; violent generalized convulsions; emesis; cyanosis; spasticity; then tremor and flaccidity.	Death
40	6	?	Same, except for prolongation of symptoms.	Death during the night
40	9	15 min.	Same.	Death
40	6	29 min.	Same.	Death
40	4.5		Same.	Death
50	2	18 min.	Same.	Death

*Normal on following morning.

TABLE IV.

Toxicity of Novocain for Guinea Pigs, Subcutaneously.

Dose in mgm. x K	Onset of symptoms in min.	Duration of symptoms	Outstanding symptoms	Result
450	5	3 hrs. 15 min.	Rear paralysis; generalized clonic and tonic convulsions with interim spasticity; episthotonus; slow, deep respirations; rapid shaking of head.	Recovery
450	10	*	Same with cyanosis.	Recovery
475	17	**	Early excitation; then same course, with tachycardia and shallow respiration.	Recovery
480	15	** 2 hrs.	Early apathy and muscular inco-ordination. Generalized convulsions—clonic and tonic. Prolonged spasticity with running movements interspersed.	Recovery
480	10	**	Same.	Recovery
500	6	3 hrs. 10 min.	Early excitation, spasticity and rear paralysis; marked episthotonus; generalized convulsions; flaccidity; cyanosis; gasping respirations.	Death
500	10	1 hr. 10 min.	Same.	Death
500	7	*	Spasticity; powerful clonic convulsions; episthotonus; cyanosis.	Recovery

*Apparently normal next morning.
**Sluggish next morning.

A 3 per cent solution of cocain instead of the usual 1½ per cent caused one fatality in an early experiment. Thirty mgm. doses of a 1½ per cent solution were withstood, but 40 mgm. proved uniformly fatal. Forty mgm. of cocain per K was taken in this study as the minimal lethal dose, although 42.5 is usually accepted. Tutocain caused one death at 180 mgm. per K, two lived with an equal dose, one lived with 190, but 200 uniformly caused death. For this study, 180 mgm. per K was accepted as the minimal lethal dose. The minimal dose of novocain was 500 mgm. per K. Tutocain was thus found to be 4.5 times less toxic than cocain and 2.7 times more toxic than novocain.

Table V shows the relative toxicity of cocain, novocain and tutocain, expressed in minimal lethal doses of mgm. per K, as reported by other investigators. In this table, for purposes of comparison on an equal basis, the figures for the higher percentages of tutocain solutions were disregarded and only those for the 1½ per cent to 2 per cent solutions were compared.

TABLE V.

Comparative Toxicity of Cocain, Novocain and Tutocain

Minimal lethal doses in mgm. per K.

Drug	Cunningham	Schuleman	Watson-Williams	Hirsch	Wagner	Chadbowne	Eggleston & Thatcher	Grode	LeBroq	Impens	Smitz & Lavenhart	Av.	Inverse toxicity ratio Cocain-1
						Guinea Pigs (subcutaneous)							
Cocain	40	0.5%—169 1 %—141			50		40					43	1
Novocain	500	2 %—126	10%—171									500	11
Tutocain	180	5 %—120 10 %—116	2%—340 1%—1000		200						For 1.5-2% sol.	11.5	4.9
						Rabbits (intravenous)							
Cocain		7-10										8.5	1
Novocain		30-40										35	4
Tutocain		15-30										22.5	2
						Rabbits (subcutaneous)							
Cocain						200	60	87-100	153-192	100-150	90	131.7	1
Novocain							730				600	665	4.5
						Mice (subcutaneous)							
Cocain											150	150	1
Novocain											900	900	6
						Rats (subcutaneous)							
Cocain											250	250	1
Novocain											1600	1600	6.4
						Cats (subcutaneous)							
Cocain							40				30	35	1
Novocain											450	450	13
						General average							
Cocain													1
Novocain													7.4
Tutocain													3.45

Guinea pigs require so great a quantity of a 1 per cent solution of tutocain to furnish the toxic dosage of this drug, that the absorption is much retarded and the result therefore not representative. For example, Hirsch finds that a dose of 1000 mgm. per K of such a solution is tolerated. The results are calculated as inverse ratios of toxicity using cocain as unity. The average of these show tutocain to be 3.5 times less toxic than cocain and about twice as novocain.

CORNEAL ANESTHESIA.

The penetration of stratified squamous epithelium of the cornea and the disappearance of the drug by absorption and lachrymation make it the favorite area for laboratory tests of surface anesthesia. The criterion for anesthesia has long been the disappearance of the winking reflex to touch. Because this sign is faulty, other methods of testing were sought.

A second test, the absence of local reaction to irritants as a criterion of anesthesia, was tried. Dionin, chloroform water and ether water were used in the eyes of rabbits anesthetized with cocain and tutocain. In the cocainized eye, the reaction was only about 25 per cent of that of the noncocainized eyes. But in the eyes anesthetized with tutocain there was no perceptbile difference between the dionin reaction in the anesthetized and nonanesthetized eyes. This shows that the two drugs are not comparable, presumably because cocain is a vasoconstrictor and tutocain is not. The dionin test is not a test of anesthesia alone, but is also a test of the relative powers of a vasoconstrictor (cocain) and a vasodilator (dionin). The test was therefore abandoned for the standard touch-wink reflex.

The technique used was that described and used by Sollman.[7] Our observations were made every two to three minutes, rather than at ten-minute intervals.

White rabbits were placed in closely-fitting stalls with their heads free and eyelashes clipped. The lower lide was pinched into a pocket, elevated and the anesthetic solution was inserted with an eye dropper. The entire conjunctival sac was flooded for one minute and the lid released. In a dim light, every two or three minutes, the periphery of the cornea was gently touched with a small transparent glass rod, bent at right angles and terminating in a smooth ball. Great care was taken not to touch the lid margins. If a wink appeared with the first light touch, the test was called "no anesthesia" and if the wink were sluggish or appeared only after fairly severe, stroking pressure, it was called "partial anesthesia." If after ten minutes the anesthesia was absent or only partial, the conjunctival sac was again momentarily flooded with the solution. This was repeated every ten minutes

until four applications were made, if complete anesthesia had not been obtained. No applications were made after anesthesia was complete. Observation was continued until sensation had returned as completely as in the opposite eye used for control. Only cocain was used for comparison with tutocain since novocain, an excellent anesthetic in contact with nerves, is very weak when applied to membranes, and is not used generally for surface anesthesia.

Table VI represents the results for tutocain, and Table VII for cocain.

TABLE VI.

Corneal Anesthesia of Tutocain.

%	No. cases	Av. no. applications	Av. onset of anes. in min.	Av. duration of anes.	Degree of anes.	Remarks
1/32	9	4			None	Possible slight lessening of sensation.
1/24	6	4			None	Possible slight lessening of sensation.
1/12	12	4	40	5.5	In 9, none; In 3, partial	Slight vasodilation.
1/8	13	2.1	13.9	9.5	Complete	Slight vasodilation. No apparent injury.
5	10	1	4.5	61.1	Complete	Vasodilation; drying of cornea; some lid-lag.

TABLE VII.

Corneal Anesthesia of Cocain.

%	No. cases	Av. no. applications	Av. onset of anes. in min.	Av. duration of anes.	Degree of anes.	Remarks
4	1	1	2	—	Complete	Mydriasis; crenation of conjunctiva and dryness.
1/2	7	1.1	6.5	15.7	Complete	Same, less drying.
1/5	16	1.5	7.8	12	In 15, complete; in 1, partial.	Same.
1/8	7	3.6	6	10	In 2, complete; in 7, partial.	

The minimal percentage for corneal anesthesia for tutocain was found to be 1/8 per cent, and for cocain, 1/5 per cent.

Table VIII shows the minimal effective concentrations found by other investigators, all using essentially the same technique, though evidently with variable interpretation of results.

It will be noted that Wagner reports the surprising figure of 1/33 per cent for the minimal concentration effect for tutocain which gave anesthesia for thirty seconds. The number of cases is not stated. His figures are included in the table of ratios (Table IX) of the comparative minimal effective concentration for corneal anesthesia, but it is felt that the ratio found in this study, which would give tutocain .625 and cocain 1, more nearly represents the relative surface anesthetic power of the two drugs. That is, tutocain appears to be about 3/5 more powerful as a surface anesthetic than cocain, measured by the minimal effective concentration alone. However, tutocain requires a somewhat longer time to produce complete anesthesia.

TABLE VIII.

Minimal Effective Percentage Solutions for Corneal Anesthesia of Cocain, Novocain and Tutocain.

	Schuleman	Sollman	Smitz and Lavenhart	Hoffman and Kochman	Wagner	Aver.
Cocain	1/20	1/2		1/2 (cir.)	1/5	1/3
Novocain	2	8	2	8 (cir.)		5
Tutocain	1/8				1/33	1/20

TABLE IX.

Anesthetic Ratios (Cocain=1).

	Other Investigators	Cunningham
Cocain	1	1
Novocain	17	
Tutocain	.34	.625

A third line of laboratory investigation—one which was not used in this study—is that of intracutaneous infiltration of dilute solutions. A wheal from 4 to 10 m.m. in diameter is made in the skin on the ulnar surface of the forearm. Testing the sense of touch by means of a cotton-tipped probe, observations are made to find the lowest percentage giving anesthesia, the time of onset, and the duration and degree of anesthesia. This method combines nerve-end and conduction anesthesia, and appears more nearly the clinical approach. In Table X are given the minimal effective concentrations for anesthesia by this method as well as the calculated anesthetic ratios for tutocain and cocain, figuring cocain as unity, according to Sollman,[8] Hoffman and Kochman,[3] and for cocain and tutocain according to Wagner.[10] The percentages are surprisingly small, leading us to suspect that the anesthesia was partly from pressure, since no controls were mentioned. The necessary concentration for anesthesia, accord-

ing to Wagner,[10] was four times less for tutocain than for cocain. He found the same ratio for corneal anesthesia.

TABLE X.

Minimal Effective Percentage Solutions for Intracutaneous Anesthesia.

	Sollman	Hoffman and Kochman	Wagner	Aver.	Direct Ratio Cocain=1
Cocain	.031	.012	.0125	.018	1
Novocain	.031	.025	.0031	.028	1.54
Tutocain				.0031	.17

A favorite method of exhibiting the merits of local anesthetics in a comparative way is to compute the ratio of the fatal dose (in mgm. per K) to the minimal effective concentration (in per cent) for surface or intradermal anesthesia. Expressing such a ratio as a fraction, the more valuable the anesthetic, the greater is the numerator and the less the denominator. Using the figures for the minimal lethal doses and minimal effective percentages for corneal anesthesia found in this study, we arrive at the series of ratios shown in Table XI. The average minimal effective concentration of novocain for corneal anesthesia found by various investigators is 5 per cent. This figure is used in the table, since in this study no attempt was made to determine this value. Tutocain thus is shown to compare favorably with novocain and cocain, but for reasons mentioned above it is manifestly unfair to compare novocain with true surface anesthetics.

TABLE XI.

Anesthetic Ratios, $\frac{\text{Minimal effective concentration}}{\text{Fatal dose in mgm. x K}}$

Cocain	$\frac{.04}{.2} = .02$
Novocain	$\frac{.5}{5.0} = .1$
Tutocain	$\frac{.18}{.125} = 1.44$

Laboratory attempts have been made to add to tutocain certain drugs which would potentiate its anesthesia by synergistic action beyond that accounted for by the simple arithmetic summation of their combined effects. Wagner[10] found in intracutaneous wheal experiments that adrenalin would prolong the anesthesia nineteen minutes but would not permit a lessening of the amount of tutocain. This was also demonstrated by Sollman.[8] Wagner and Ghese[10] found

that an added .4 per cent of potassium sulphate gave a true potentiation and an earlier onset with intracutaneous injections. Sollman[8] secured only a summation effect by addition of potassium sulphate and concluded from the variations in his own and other experimenters' results that too much should not be expected of such mixtures.

Wagner[10] found the surface anesthetic power of tutocain was enhanced two or three times and that of cocain one-and-one-half to three times by the addition of .35 per cent phenol.

The only mixture prepared for general distribution is that of adrenalin with tutocain.

CLINICAL REPORTS.

The effects of tutocain upon tissues from the practical operative standpoint were tested in a series of fifty cases distributed as follows: Tonsillectomies, *35*; removal of middle turbinate and ethmoids and sphenoids, *6*; submucous resections, *5*; and antromeatal operations, *4*.

Certain routine methods were used. Each patient was given a hypodermic dose of morphin and atropin 45 to 60 minutes preoperative. Each was operated in the sitting position. The time of the appearance of anesthesia was determined by frequent probing. The patient was requested to avoid swallowing any of the anesthetic solution and to report at once any toxic symptoms or pain. The pulse was taken five or six times during each operation, and the patient was kept under constant observation during and after the operation.

Five per cent tutocain was applied for surface anesthesia and 1/5 per cent was injected for infiltration. Adrenalin, 1:10,000 in sterile saline was used with the 5 per cent solution for surface anesthesia and from three to six applications were made, brushing freely with a cotton-tipped applicator the pharyngeal mucosa or, in the nose, the regions of the sphenopalatine ganglia and nasal nerves.

Twelve c.c. of a 1/5 per cent solution, containing .06 mgm. adrenalin, were used for injection. The spaces between pillars and tonsil were infiltrated in the usual manner. For a submucous operation minums 5 to 7 of additional adrenalin 1:1000 were injected along the site of incision. Light surface anesthesia in the throat always appeared in from three to five minutes, usually with disappearance of the gagging reflex, and within ten minutes from the time of the first application the patient was ready for operation. If the injection were finished before this time it was found best to wait two minutes longer. If this extra time were allowed no case was found in which the anesthesia was not as satisfactory and as complete as with the 10 per cent cocain and .5 per cent novocain used in this department

for tonsillectomy or the cocain flakes and adrenalin mixture used for intranasal operations.

Since tutocain is not a vasoconstrictor, bleeding was satisfactorily controlled in tonsil work and fairly well controlled in intranasal sinus operations by the use of adrenalin as noted above, although but half the amount was used with tutocain as with novocain. In submucous resections, however, in spite of the free use of adrenalin, several of the operations were more bloody than with the usual cocain flakes and adrenalin and were prolonged by the necessary sponging.

The duration of the anesthesia, although not exactly determined, was always sufficient for operative work. The longest operation—a submucous resection—lasted forty minutes and was not attended by unusual discomfort.

Tutocain was especially satisfactory from the viewpoint of the absence of toxic by-effects. Except in two cases, noted below, the color remained normal and the patients persistently denied any subjective toxic symptoms. The pulse tended to rise at first in the operating room and during the injection of the tutocain and adrenalin. The average maximum pulse rate was 103. During the operation the rate usually remained from 80 to 90.

One young woman just after the injection had a transient feeling of faintness. Her face grew pale but her lips remained normal in color. Her pulse showed a transient irregularity, but remained fairly strong and 80 to the minute.

A second patient, a man age 48 years, complained of dark spots before the eyes and a feeling of faintness twelve minutes after the beginning of five applications of the 5 per cent solution in 1:10,000 adrenalin to the nasal mucosa for removal of the left middle turbinate and polypi, and eviceration of ethmoids and sphenoids. His face grew somewhat pale, the pulse rate dropped to 50, but the pupils did not dilate. Lowering the head at the time and again four minutes later, when the faintness reappeared, gave immediate relief, and the pulse resumed the rate of 80.

The symptoms in both the above cases can hardly be ascribed to adrenalin, and since the pulse rate remained normal or subnormal they were probably psychic in nature.

The only death from tutocain anesthesia which has been found in the literature is that reported by Schwartz.[44] The symptoms were unconsciousness, clonic convulsions and cyanosis, followed by death in forty minutes after a urethral injection of 8 c.c. of a 2 per cent solution, or .16 gm. of tutocain. The autopsy diagnosis was status lymphaticus, and death was considered to be due to poisoning from a dose usually harmless.

Smaller dosages of local anesthetics must be used about the head and neck than elsewhere in the body. Arithmetically considered on a basis of the animal toxicity figures quoted above, however, the amount of tutocain used is far within the limits of safety. It is difficult to estimate the amount of the 5 per cent tutocain in 1:10,000 adrenalin solution which is absorbed from the local application, but it must necessarily be small. Twelve c.c. of 1/5 per cent solution contains 24 mgm. of tutocain and .06 mgm. of adrenalin. We have never seen toxicity from this amount of .5 per cent novocain (60 mgm.) and .125 mgm. of adrenalin injected about the throat. Considering tutocain 2.7 times as toxic as novocain, according to the guinea pig experiments made in this study, the toxicity of the doses of tutocain and novocain is practically the same. The adrenalin dosage used in combination was half that usually used with novocain in this department, not considering the amount absorbed from the topical application.

In proportion to weight, the minimal lethal dose of 180 mgm. per K found for guinea pigs would require for a man of 65 K 11.7 grams for a lethal dose, if the toxicity for man and guinea pig were proportionate, or 479 times the amount used in a single operation.

The effect of tutocain upon the blood pressure was not studied. Schuleman[5] found it depressed in rabbits for a short time after the administration of convulsive doses of tutocain.

Hirsch[27] considers suprarenin indispensable in the use of tutocain as a surface anesthetic. He has observed that its power is greatly increased when employed in a .5 per cent phenol solution for topical application, and with adrenalin combined with .4 per cent potassium sulphate for infiltration. When used upon the larynx and bronchi, a 5 per cent solution was found sufficient, and it appeared to be superior to cocain. This did not seem to be always the case in its nasal application. He concludes that the anesthetic power of tutocain is greater than that of any other synthetic local anesthetic, and that it is better adapted for both surface application and for injection.

P. Watson-Williams and E. Watson-Williams[11] present their conclusions regarding tutocain from its use in seventy operations and numerous other observations. They found a 5 per cent solution of tutocain to give clinically the same result as 3.5 per cent cocain, the difference being due to a delayed action on the part of tutocain rather than to its failure to give the ultimate effect. They controlled bleeding by the addition of adrenalin 1:10,000. No toxic by-effects were noted.

H. Czermak[26] used 3 per cent tutocain for surface anesthesia in examinations and 10 per cent for local operations and endoscopic

examinations. He found anesthesia with tutocain to be slower but just as complete as with cocain. A .5 per cent solution was satisfactorily used in operations upon the middle ear and antrum. No toxic by-effects were observed. He concludes that tutocain closely approximates cocain in value, with the advantage that it is much less toxic, and is the best available anesthetic for injection, being superior in his judgment to novocain.

Seifert and Anthon[30] concluded after one-and-one-half years of use for surface and injection anesthesia, that .25 per cent tutocain was the equal of .5 per cent novocain and that it was adapted for intranasal work, tonsillectomy, tracheotomy, laryngectomy, esophagotomy, strumestomy, operations on the ear and various kinds of plastic work. A 1/8 per cent solution was found satisfactory if a sufficient amount of the solution were used. They employed a 10 per cent tutocain-adrenalin solution for ordinary surface anesthesia and 20 per cent for bronchoscopy or esophagoscopy. They found these equivalent to the same concentrations of cocain in the degree of anesthesia produced, the tutocain anesthesia being somewhat slower. They encountered no toxic by-effects.

Sybrecht[32] used eight drops of a 5 per cent solution with from 1 to 2 drops of 1:1,000 adrenalin solution, making from four to six applications for surface anesthesia. He found the anesthesia lasted from forty to sixty minutes, but encountered more bleeding than with cocain.

Riedel[29] concludes that tutocain can largely replace cocain in rhinolaryngology. His patients were ready for operation ten to fifteen minutes after the application of the 5 per cent tutocain, which he considered to be equivalent to 10 per cent cocain if additional time were allowed for the tutocain to act. He injected ⅛ to ¼ per cent solution and reports no unpleasant by-effects.

Suchanek[31] used tutocain for surface infiltration and conduction anesthesia in 300 cases. He used 5 per cent solution for surface anesthesia, ⅛ per cent for infiltration, and 1/5 per cent for conduction. He reported twenty-four bronchoscopies and esophagoscopies and five endolaryngeal operations as successful under tutocain anesthesia. For intranasal operations Hirsch's mixture (tutocain, adrenalin and potassium sulphate) proved satisfactory. He noted no toxic by-effect and concludes that tutocain is superior to cocain in efficiency and in its lower degree of toxicity. He ascribes to it a slight hyperemic effect.

CONCLUSIONS.

I. Animal experimentation with tutocain, as compared with cocain and novocain, shows:

1. It is 4.5 times less toxic than cocain.

2. It is 2.7 times more toxic than novocain.

3. It is approximately 3/5 more powerful than cocain for surface anesthesia.

4. The toxic symptoms are essentially those of cocain and novocain.

II. Comparative clinical use of tutocain shows:

1. A 5 per cent solution in 1:10.000 adrenalin is a satisfactory substitute for 10 per cent cocain for the surface anesthesia in the removal of tonsils.

2. A 1/5 per cent solution is a satisfactory substitute for .5 per cent novocain for infiltration in the removal of tonsils.

3. A 5 per cent solution in 1:10,000 adrenalin is a satisfactory substitute for flake cocain and adrenalin in most intranasal operations.

4. It is less satisfactory than cocain flakes and adrenalin for submucous resections because of the tendency to bleed.

5. It is somewhat slower than cocain and novocain in producing equivalent anesthesia.

6. It is superior to cocain in its relative freedom from toxic by-effects.

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University of Iowa.

A NEW TONSIL EXPRESSOR.*

DR. A. LOBELL, New York.

We have two ways to determine the presence of pus within the tonsils. One is suction, and the other is pressure. The latter is more commonly employed, perhaps on account of its simplicity. It is accomplished by forcing a tongue depressor against the anterior pillars. This invariably results in a certain amount of trauma because a narrow blade is bound to injure a delicate structure. It causes pain and discomfort and sometimes a nervous patient is scared of a little bloody expectoration that follows. Owing to these obstacles a complete search for pus is often abandoned by this method.

THE AUTHOR'S TONSIL EXPRESSOR.

With the object to eliminate most of the unpleasant features of the old method, the new tonsil expressor was devised by the author. It consists of a shaft joined to an appropriate handle. The shaft divides into two prongs. A pin running through the transverse axis

of the steel ball is fastened to the prongs at their distal ends. The perforation in the ball is somewhat larger than the pin, permitting the ball to revolve very easily. The instrument is made in two sizes. One carries a ½-inch and the other a ⅜-inch ball. The smaller size is valuable in small and buried tonsils.

Technique: A tongue depressor in one hand pushes the tongue out of the tonsillar field and the instrument is held in the other hand. The ball is firmly applied to the upper pole of the anterior pillar and it is rolled downward slowly until the lower end of the pillar is reached. The same manoeuver is now reversed from below upward. In the majority of instances this will suffice to find pus when it is present. However, if the results are not convincing, the procedure can be repeated without fear of any injury. The writer has employed this instrument at the Manhattan Eye, Ear and Throat Hospital, at the Lebanon Hospital Clinic and in private practice

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for the last nine months on about five hundred patients, at least, with gratifying results and without any untoward complaints.

Indications: It is self evident that its usefulness is mostly exhibited as a diagnostic procedure. The writer wishes, however, to point out that in a small number of cases where tonsillectomy was contraindicated and there was considerable pus within the tonsils, the procedure was found satisfactory as a palliative therapeutic measure. The patients experienced local and general relief after pus was evacuated. Whenever expression was resorted to for palliative purposes, the following steps were found convenient:

1. Swabbing the tonsils with 10 per cent cocain.
2. Probing apparently clogged crypts.
3. Tonsillar expression as described above.
4. Clearing away the pus by suction, gargle or sponge.
5. Introduction of colloidal silver solution on a cotton wound applicator into the crypts.

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